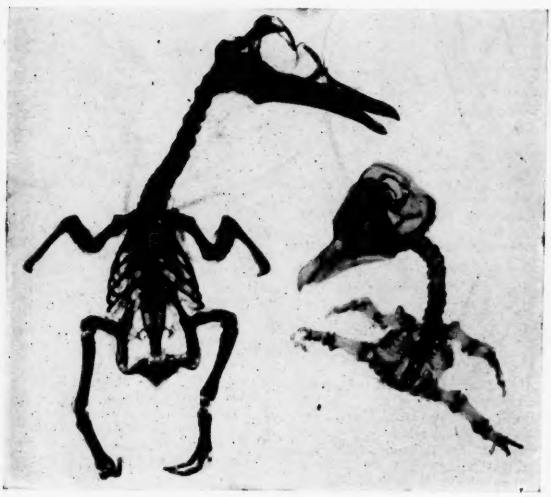
The Journal of HEREDITY



GENE-STUNTED PIGEON
(SEE PAGE 297)

Rare Rh Alleles

Bud Mutation in the Marigold

Designating Multiple Births

Ancient Chinese Note on Heredity

News of Dutch Geneticists

Partial Sex-Linkage of Webbed Toes

VOLUME 36 OCTOBER, 1945 NUMBER 10

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

DAVID FAIRCHILD, President — Vice-President, SEWALL WRIGHT — Secretary, C. E. LEIGHTY
Treasurer, SAMUEL L. EMSWELLER

Managing Editor, ROBERT C. COOK

Business Manager, NANCY KING

EDITORIAL BOARD

M. DEMEREC — RALPH W. PHILLIPS
H. J. MULLER — R. R. GRAVES — PAUL POPENOE

Members of the Council are ex-officio members of the Executive Board

COUNCIL

O. S. AAMODT, Head Agronomist, Division of Forage Crops and Diseases, Plant Industry Station, Beltsville, Md.
RONALD BAMFORD, Professor of Botany, University of Maryland, College Park, Md.
W. E. CASTLE, Emeritus Professor of Genetics, Harvard University, and Research Associate of the Carnegie Institution of Washington.
LEON J. COLE, Professor of Genetics, University of Wisconsin.
O. F. COOK, Bionomist (Retired), U. S. Department of Agriculture.
GEORGE M. DARROW, Horticulturist in Charge of Small-Fruit Breeding, Horticultural Field Station, Beltsville, Maryland.
DAVID FAIRCHILD, Agricultural Explorer (Retired), U. S. Department of Agriculture.
W. V. LAMBERT, Assistant Research Administrator, Agricultural Research Administration, U. S. Department of Agriculture.
C. E. LEIGHTY, Agronomist in Charge of Dry Land Agriculture, U. S. Department of Agriculture.
HUGH C. MoPHEE, Chief of Animal Husbandry Division, U. S. Department of Agriculture, Washington, D. C.
MARCUS M. RHOADES, Department of Botany Columbia University, New York City.
SEWALL WRIGHT, Professor of Zoology, University of Chicago.

ADVISORY COMMITTEE

W. S. ANDERSON, Genetics Department, University of Kentucky, Lexington, Kentucky.
E. B. BABCOCK, Genetics Department, University of California, Berkeley, California.
E. A. BESSEY, Professor of Botany, Michigan State College, Lansing, Michigan.
A. F. BLAKESLEE, Professor of Botany, Smith College, Northampton, Mass.
W. E. BRYAN, Professor of Plant Breeding, University of Arizona, Tucson, Arizona.
RUSSELL W. DUCK, Editor, *The Rural New Yorker*. New York City.
R. A. EMERSON, Professor of Plant Breeding, Cornell University, Ithaca, New York.
C. P. GILLETTE, President, The Genetic Foundation, Fort Collins, Colorado.
C. M. GOETHE, Sacramento, California.
HARRISON B. HUNT, Professor of Zoology, Michigan State College, East Lansing.
H. S. JENNINGS, Emeritus Professor of Zoology, Johns Hopkins University, (Berkeley, Calif.).
D. F. JONES, Plant Breeder, Connecticut Agricultural Experiment Station, New Haven, Conn.
M. A. JULL, Professor of Poultry Husbandry, University of Maryland, College Park, Md.
F. B. LINFIELD, Director, Montana Experiment Station, Bozeman, Montana.
C. C. LITTLE, Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.
JAY L. LUSH, Professor in Animal Husbandry, Iowa State College, Ames, Iowa.
H. H. NEWMAN, Emeritus Professor of Zoology, University of Chicago, (Clearwater, Fla.).
T. S. PALMER, Expert in Game Conservation, U. S. Dept. of Agriculture, Washington, D. C.
JOHN H. PARKER, Director, Kansas Wheat Improvement Association, Manhattan, Kansas.
JOHN W. SCOTT, Professor of Zoology, University of Wyoming, Laramie, Wyoming.
A. D. SHAMEL, Physiologist, U. S. Department of Agriculture, Riverside, California.
GEORGE H. SHULL, Professor of Biology, Princeton University, Princeton, New Jersey.
EDMUND W. SINNOTT, Professor of Botany, Yale University, New Haven, Conn.
W. W. SMITH, Professor of Animal Husbandry, Purdue University, Lafayette, Indiana.
A. B. STOUT, Director of Laboratories, New York Botanical Gardens, New York City.
W. T. SWINGLE, Plant Physiologist (Retired), U. S. Department of Agriculture.
W. H. TOMHAVE, American Aberdeen-Angus Breeders' Association, Chicago, Ill.
H. E. WALTER, Professor of Biology, Brown University, Providence, Rhode Island.
H. J. WEBBER, Prof. Subtropical Hort., Citrus Experiment Station, Riverside, California.
E. N. WENTWORTH, Armours' Bureau of Agr'l Research and Economics, Chicago, Illinois.
P. W. WHITING, Zoological Laboratory, University of Pennsylvania, Philadelphia, Pa.

The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore, Md., and 1507 M St., N. W., Washington, D. C. Address all communications regarding editorial matters to 1507 M. St., N. W., Washington, D. C. Communications regarding the business management of THE JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to 1507 M St., N. W., Washington 5, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 29, 1918.

Contents copyrighted 1945 by the American Genetic Association. Unauthorized reproduction of material from THE JOURNAL not permitted. The title "Journal of Heredity" is registered as a trade-mark in the U. S. Patent Office.

$3.50 a Year. 35c a Copy. Prices of back numbers on application.

The

Journal of Heredity

Vol. XXXVI **OCTOBER, 1945** **No. 10**

CONTENTS

BUD-MUTATIONS IN THE MARIGOLD
By Hugo Iltis 291
A LETHAL ACHONDROPLASIA IN THE PIGEON
By W. F. Hollander 297
HEREDITY OF THE RH BLOOD TYPES
By Eve B. Sonn and Alexander S. Wiener 301
INHERITANCE OF COAT COLOR IN SWINE
By H. O. Hetzer 309
TWO NEW PEDIGREES OF ZYGODACTYLY
By Alan C. Pipkin and Sarah Bedichek Pipkin 313
FUSED TEATS
By Leslie E. Johnson 317

Notes and Reviews

SYMBOLS FOR DESIGNATING MULTIPLE BIRTHS 295
ANCIENT CHINESE KNOWLEDGE OF HUMAN HEREDITY
By Richard B. Goldschmidt 296
RESEARCH IN BIOLOGY AND NUCLEAR PHYSICS 300
GENETICS IN HOLLAND 304
GENETICS AND CLINICAL PROBLEMS (Rev. of BAUER: *Constitution and Disease*)
By Laurence H. Snyder 305
POPULATION PROBLEMS (Rev. of BURCH and PENDELL: *Population Roads to Peace or War*)
By Karl Sax 306
AN ABSTRACTING SERVICE FOR HUMAN BIOLOGY 307
"WITHOUT PRIDE OF ANCESTRY" 320

STUDENT SUBSCRIPTIONS

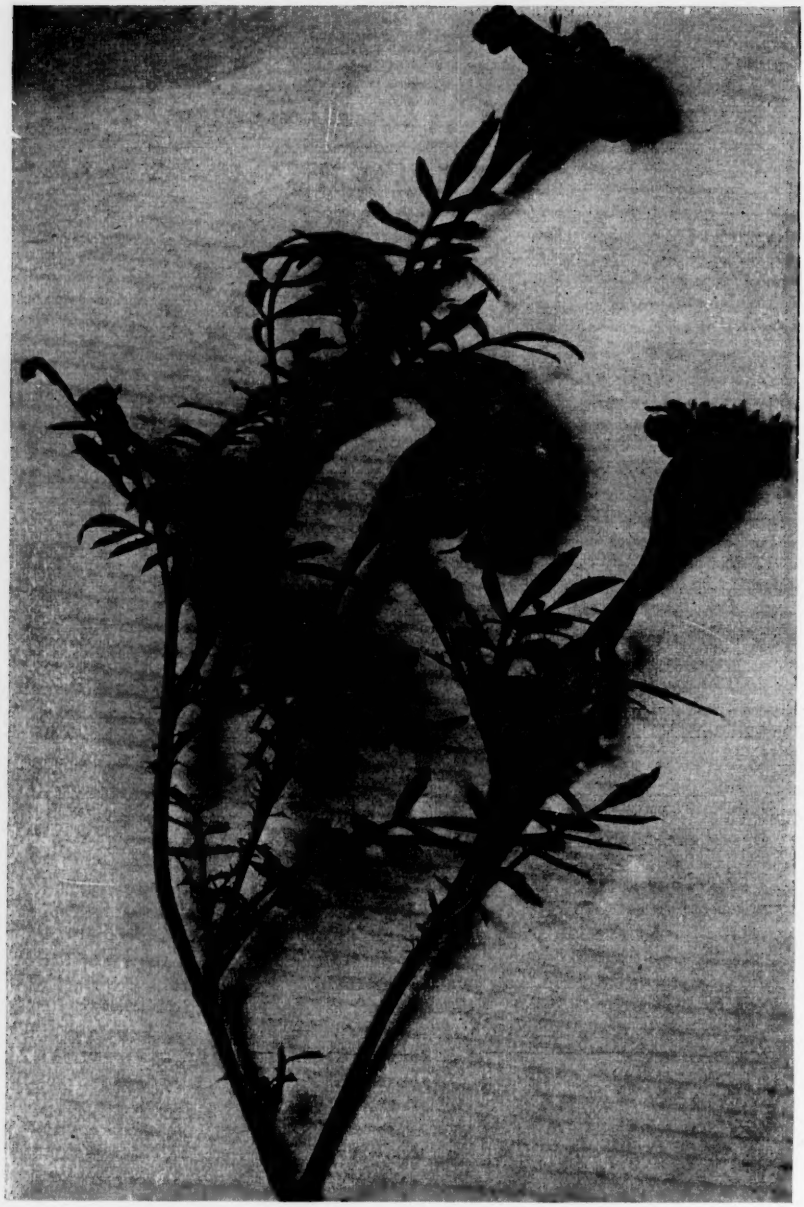
Members engaged in teaching genetics or related subjects are cordially invited to use the JOURNAL as a supplementary text in their classes. The rapid progress of the science, after being so long neglected, is one of the most important and attractive features to place before students. Even the latest textbooks are soon outdated, but they become more interesting as the historical background of the discoveries described in the JOURNAL OF HEREDITY.

Student subscriptions covering the academic year of eight months, in groups of ten or more, may be ordered by members at one dollar ($1.00) each, four months for 50 cents. These special rates to students will be accepted only when applied for by members of the Association, in whose classes the Journals are to be used. The *student subscriptions* will be sent in bulk to the member in whose name they are ordered.

Special Trial Offer to Members of A. G. A.

Members engaged in teaching will be supplied free up to twelve copies of two issues for classroom use. Try the JOURNAL for two months at no expense and under no obligation to continue. Issues for this purpose will be sent in bulk to members applying for them. Send orders to

AMERICAN GENETIC ASSOCIATION
1507 M STREET, N. W. WASHINGTON 5, D. C.



TWO KINDS OF MARIGOLD FLOWERS ON ONE PLANT

Figure 1

Two species of marigold are commonly raised in gardens in the United States. The French marigold has a single flower with a collar of petals while the African marigold has a chrysanthemum-like double flowerhead. Here both kinds of flowers are found on the same plant. Three such curious chimeras appeared among the plants grown from a single packet of seed.

BUD-MUTATIONS IN THE MARIGOLD

HUGO ILTIS

Mendel Museum, Fredericksburg, Va.

CHARLES Darwin defined as bud-mutations "all these sudden changes in structure or appearance which occasionally occur in full-grown plants in their flower buds or leaf buds."[4] DeVries called bud-variations "the finest example of spontaneous changes. . . . The new form arises as a bud or twig on an individual of the old form and often remains a long time united with it. In a case like this there can be no doubt as to the mutual genetic relationship."[7]

My paper describes a peculiar and conspicuous bud-mutation observed simultaneously in three plants of a bed of French marigolds (*Tagetes patula*) in the fall of 1943.

Tagetes is a genus of the subfamily Heleniae of the ligulate Compositae, native to Central and South America. Some species like *T. erecta* and *T. minuta* have become completely naturalized in the southern United States.[13] Two species most commonly used in cultivation are the "African" marigold (*T. erecta*) and the "French" marigold (*T. patula*). The former is a Mexican species, characterised by a flower head composed of compact tubular or quilled "petals" making a large globe of golden yellow or orange. The "French" marigold (*Tagetes patula*) bears smaller flower heads, often two-toned in yellow and velvet brown, with flat and overlapping petals.

The most important garden varieties of *Tagetes* have been known to science for a long time. According to Sweet's *Hortus Britannicus, T. patula* was introduced into cultivation in 1573, *T. erecta* in 1536.[2] In 1629 Parkinson in his *Paradisus* mentioned the tubular form of the floret of "the single hollow French marigold." The "Chrysanthemum type" flower heads of the African marigold were illustrated in the *Plants of Mexico* by Francesco Hernandez in 1640.[11] For nearly 400 years by cultivating and crossing these and several other species, gardeners have produced a large number of commercial varieties. The genus has, however, been neglected by scientific breeders and geneticists. As far as I am aware only two short papers on the genetics of *Tagetes* have been published. One by Punnett[12] deals with the inheritance of two character-pairs, "double" *vs* "single," and "flat petals" *vs* "tubular petals." The other, published recently in this JOURNAL, investigates the genetics of the characters virescent and early blooming.[10]

In October 1943 my wife called my attention to three plants in a bed of Dwarf French marigolds in our garden exhibiting a very conspicuous and characteristic phenomenon. Each of these plants bore a number of flower-heads representing the "single French" type with about 8-9 velvet mahogany ray flowers surrounding about 15-30 bright yellow disc flowers. But beside these normal flower heads there were on each of these three plants other flowers with no or with abortive petaloid ray flowers but with a very much increased number (about 90) of bright yellow disc-flowers. This second type resembles the "chrysanthemum" type of the African marigold of the flower catalogues. The difference between the two types was very conspicuous. The first of these plants as shown by our photograph had three "single French" and two "chrysanthemum" flower heads. The second had four "single" and two "chrysanthemum" heads and the third had three heads, two "single" and one "chrysanthemum." Altogether there were eight "single" and five "chrysanthemum" flower heads produced by the three plants.

The plants were raised from a seed package of "Dwarf French marigold"

ALTERNATE FRENCH AND AFRICAN MARIGOLD FLOWERS

Figure 2

Of the five flowers on this plant three are French marigolds and two are typical of the African species. The varietal names French and African are really misnomers, because the marigold is native to Central and South America. It is known that in some crosses the single French type of flower is recessive to the chrysanthemum-like double flower. Possibly these plants are hybrids, and through abnormal chromosome distribution the recessive type has become manifest. Why this should occur in three plants in a relatively small population is not known.

bought for ten cents in a department store. The phenomenon was evidently a "bud-mutation" since flower heads of completely different types appeared together on the same plant. Since the bud-mutations were produced simultaneously by three different plants in the same bed they probably resulted from the conditions of the environment acting on a peculiar genotype.

The analysis of the flower heads gave the following results. The *"single French" flower heads* have a single cylindrical involucre with light bracts and six to nine well developed, flat, strap-shaped petaloid ray flowers with golden brown velvet perianths. All of them are pistillate with a style, two stigmas and a well developed ovary. They include a small number (15-30) of tubular bright yellow hermaphroditic disc flowers with four to six petals and a tube of anthers around the style. At the time they were dissected most of the flowers were immature and showed a rather small ovary, crowned by two transparent scales representing the pappus.

The *"chrysanthemum" type flower heads* were characterized by a conspicuous increase in the number of yellow disc flowers and by a decrease in the number and development of the ray flowers. They showed only four or five inconspicuous ray flowers which, at the time of the examination, were wilted but which had well developed ovaries. The rest of the inflorescence was composed of a large number (50-90) tubular yellow disc flowers, each having five or six or even seven petals. Most of them had six petals and three stigmas (sometimes two) above the anther-tube, and small ovaries crowned by two transparent scales. The appearance of both kinds of flower heads seemed to be quite normal except that in some of those of the "chrysanthemum" type the involucre was bursting, probably because of the pressure of the large number of disc flowers. This phenomenon is not uncommon in some double varieties of flowers, such as carnations.

I sent photographs and dried plants with a letter to Mr. David Burpee, president of the W. A. Burpee Seed Co., well known for his cultures of marigold. In his answer he wrote: ". . . For the past ten years marigolds have been my principal hobby and I must confess I have never seen anything like yours. It is especially interesting to know that you found three plants showing the same bud mutation in your garden this year. . . . From the photographs the African type flower would appear to be what we call Chrysanthemum-flowered such as Early Sunshine type and the single French flower resembles an old and practically obsolete variety known as Josephine. . . . I hope that you have been able to save some seed from both the single and the double flowers and that you will grow these next summer. . . ."

Unfortunately two of the three plants displaying the phenomenon were killed by an early frost. Only the one plant I potted and kept in the house was saved. From this plant I was able to obtain a few seeds. From the ray flowers of the "single French" type 15 achenes were produced. The disc flowers remained sterile. From the "chrysanthemum" type seven fruits were gathered all of them produced by the ray flowers. Although the ray flowers of this type showed only a "vestigial" perianth they seem to be the only ones which were fertile. From the numerous disc flowers no seeds were obtained.

Since the seeds seemed to be very light and feeble I asked Mr. Burpee to give them to one of his experienced gardeners to be cared for. But in spite of all precautions only one of the seeds germinated, producing a very weak plant. This one plant was given special attention, but died before it was big enough to be transplanted. Possibly the lack of viability of the seeds was connected with a disturbance of the genetic equilibrium in the sex cells. Consequently it has been impossible to obtain any information by crossing experiments.

Discussion

Bud variations have long challenged the interest of biologists. Darwin devoted much space to this subject in his classical study of variations.[4] De Vries[7] considered them as important supporting evidence to his mutation theory of evolution. Darwin suggested that plants producing such variations might be hybrids, a point which may be germane to an explanation of the perplexing case we are reporting.

Most of these early discussions were speculations based on general observations without knowledge of the genotype of the material or of the cytological situation within the nucleus. Darwin's term "autogenous chimeras" is still used by modern geneticists; they "originate in somatic cells as a result of some natural process by which diverse cell lineages arise from certain individual cells."[4] But most of this labelling of the end result as "spontaneous" and "autogenous" has been only a polite expression for a lack of knowledge of the internal or external conditions responsible for the phenomena.

Whether the development of the two different kinds of flowerheads on our *Tagetes* plants depends on one allelic pair or on a group of genes is not known. Punnett writes "the observation given above suggests that in the African Marigold the single form is recessive to the double."[12] The tendency which changes the dominant ligular flowers of the double into the recessive tubular flowers of the single continues to work also in our two types changing the remaining ligular flowers of the single French into the tubular flowers of the chrysanthemum type. We may therefore assume that in our case the chrysanthemum type is recessive to the single French type. Then the bud-mutation could be explained by a change in dominance: the recessive chrysanthemum character of the heterzoygous plants became manifest under the influence of extreme and exciting conditions.

The *Tagetes* plants grown in my garden were of unknown genotypes. The seeds in the cheap seed packages are often harvested from beds which were exposed unintentionally to cross-fertilization. Thus the possibility exists that they may have been heterozygous. The conditions under which the marigold plants were growing in my garden were abnormal and extreme. They were planted on the west side of the wall of our garage in very poor soil and exposed to the rays of the scorching afternoon sun. The summer of 1943 was hot and dry. Since we were absent from home for about five weeks the plants received no care, even to the extent of watering. It may be that this "hunger and thirst situation" has provided the condition which by disturbing the genetic equilibrium caused the change of dominance in some of the flower heads and therefore the described bud-mutations.

Some progress in an understanding of the origin of at least some bud-mutations has been made, however. The mutation rate of different genes has been studied in such diverse organisms as maize, delphinium and Drosophila. Demerec's work with two mutable genes of delphinium may have a bearing on this problem.[5] Both these genes mutated from "rose" and "lavender" to purple. One of them was subject to genetic change only at a specific stage late in ontogeny, producing pink flowers with small purple spots. The other gene tended to mutate at any stage during development, and the resulting islands of mutant tissue might involve either a half of an inflorescence or a sector of a petal. Polyploid chimeras have been experimentally produced by Blakeslee and others.

The cases described by Demerec and Blakeslee are justly regarded as periclinal chimeras,[14] since the normal and the mutated characters depend on a mere morphologic difference between epidermal tissues (thorny and thornless blackberry, Blakeslee) or on a difference of the pigment content in the epidermal and subepidermal layer (rose and lavendar to purple petals of del-

phinium, Demerec[5]). The mutation from French to African marigolds, however, is characterized by an increase of tubular disc flowers and the obliteration of the petals of the ray flowers, that means by differences concerning *all tissues* and not only the epidermal and subepidermal layers. It therefore cannot be a periclinal chimera, but could be regarded as sectorial.[14] Some resemblance to our case may be found, perhaps, in Nawashin's sectorial chimera of *Crepis tectorum* (see photo in Hurst's *"The Mechanism of Creative Evolution"*).[8] One half of that plant was proved to be trisomic, while the other half showed the normal diploid chromosome number resulting from the elimination of the triplicate *B*-chromosome. We may apply a similar explanation to our case. The mother plant could have been a hybrid, originating from a cross between a tetraploid and a diploid race of French marigolds and producing an unbalanced trisomic race. The African flower heads appearing on the French plants were the result of bud-mutations eliminating the extra chromosome thus restoring the genic balance. Since all seeds of the hybrid would inherit not only the trisomic constitution but also the tendency to restore the balance in some of their vegetative cells by bud-mutations, the simultaneous appearance of three plants exhibiting the same mutation could be understood. Of course, this explanation is only hypothetic and not proved by cytologic investigation.

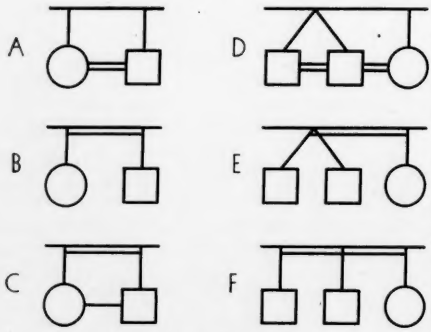
The tendency of trisomics to produce bud-mutations may have been evoked by the extremely abnormal environmental conditions, the "hunger and thirst situation" under which the plants were growing. That temperature and drought can be responsible for developing of irregularities in the number and behavior of the chromosomes has been shown by De Mol's experiments with tulips and hyacinths and by Bleier's experiments with wheat plants subjected to high and low temperatures.[6]

Literature Cited

1. Babcock, E. B., and R. E. Clausen. Genetics in Relation to Agriculture. 1937. p. 363.
2. Bailey, L. H. The Standard Cyclopedia of Horticulture. Vol. IV. 1943.
3. Darrow, G. M. *Jour. Hered.* 1926. p. 409.
4. Darwin, Ch. The Variation of Animals and Plants under Domestication. New Ed. 1896. pp. 397, 401, 439, 440.
5. Demerec, M. *Jour. Hered.* 1933. p. 369.
6. De Mol, W. E. *Zeit. f. ind. Abst. and Vererb.* 1930. p. 303.
7. De Vries, H. The Mutation Theory. Chicago. 1909. Vol. I. p. 53. Vol. II. p. 82, 184.
8. Hurst, C. C. The Mechanism of Creative Evolution. Cambridge. 1933. p. 97.
9. Iltis, H. *Zeit. f. ind. Abst. und Ver.* 1911.
10. Little, T. M., T. H. Kantor, and B. A. Robinson. *Jour. Hered.* 1940. p. 73.
11. Little, T. M. Letter to the author.
12. Punnett, R. C. *Studia Mendeliana* (edited by H. Iltis). Brno, 1924. pp. 185—.
13. Small, T. K. Manual of the Southeastern States. New York. 1933.
14. Winkler, H. Untersuchungen ueber Pfropfbastarde. Jena. 1912.

SYMBOLS FOR DESIGNATING MULTIPLE BIRTHS

EFFORTS to improve existing symbols as used in genetic pedigree charts constitute progressive steps in the science of heredity. The recent article by Macklin[1] in this Journal is appreciated as a contribution to this objective. The present writer is in favor of the adoption of Dr. Macklin's improved symbols for miscarriages and stillbirths as well as of those designating cousin marriages and identical twins. It is unfortunate, however, that Dr. Macklin chose the "double bond" as representative of fraternal twins (Figure 3*A*). In the writer's opinion fraternal twinning may be symbolized equally well and without confusing the proper use of the marriage line by merely doubling the fraternity line above and between the progeny symbols for the fraternal twins (Figure 3*B*). Confusion of the marriage line in Figure 3*A* would be rare, it is true, but it is a possibility and as such it should be avoided. Although no



SYMBOLS COMPARED

Figure 3

A—Macklin's symbol for fraternal twins. *B*—The author's symbol for fraternal twins which avoids disturbing the marriage line. *C*—Symbol representing marriage of fraternal twins. *D*—Macklin's symbol for triplets comprising identical twin boys and a sister. *E*—The author's symbol for the same triplets shown in *D*. *F*—Symbol suggested for fraternal triplets; compare with *B*.

record of the marriage of twins has been discovered by the writer a brother-sister marriage in this country has been reported as recently as 1940 in the *Scientific American*.[2] It is well known that such marriages were common long ago among the royal families of Egypt and the Hawaiian Islands.[3] The employment of pedigree charts for live stock, pets, etc., might require recording matings of fraternal twins much more often than in the case of man (Figure 3*C*). This likelihood further indicates the need of caution in the selection of symbols so as to avoid both ambiguity and confusion in the use of previously accepted signs such as the marriage line.

Dr. Macklin submitted another symbol (Figure 3*D*) the adoption of which might also lead to difficulty. This designation was intended to represent triplets comprising a pair of identical twin boys and a sister. As a simplification of her symbol (Figure 3*D*) it is suggested that a different one (Figure 3*E*) be adopted. This symbol avoids confusion in the use of the marriage line. Similarly, instead of using a "double bond" between each of the siblings of a set of fraternal triplets, quadruplets, etc., the double fraternity line would again be utilized (Figure 3*E*).

The above suggestions may seem trivial, but since analysis of genetic data depends so much upon the use of proper symbols it is important that such be carefully chosen for their simplicity and freedom from ambiguity.

Literature Cited

1. Macklin, Madge T. *Jour. Hered.* 36: 222-224. 1945.
2. *Scientific American*. 162:232. April 1940.
3. Popenoe, Paul. *Hygeia*. p. 980—. Nov. 1937.

Lawrence M. Ashley
College of Medical Evangelists, Loma Linda, Calif.

◆

Ancient Chinese Knowledge of Human Heredity

In one chapter of the writings of the Chinese philosopher and mystic Chuangtse, who died around 275 B.C., the following phrase is found according to a recent translation by Lin Yutang:* "Joined toes and extra fingers (i.e. the Mendelian characters web-toe and polydactyly) seem to come from nature, yet, functionally speaking, they are superfluous." As the following phrase begins "Goiters and tumors seem to come from the body," it appears justified to interpret the words "to come from nature" as meaning: are inborn, hereditary; "nature" being used in the Spencerian sense of nature vs. nurture. The philosopher obviously quotes the observation as a generally known fact, which he uses for the sake of drawing an ethical parallel. While it would be far fetched to call Chuangtse a precursor of human genetics, yet it seems worthy of recording that he could use facts of human heredity as a matter of common knowledge when elaborating a philosophical idea by means of a simile.

Richard B. Goldschmidt
University of California, Berkeley

*The Wisdom of India and China. Edited by Lin Yutang. Random House, New York

See p. 666.

LETHAL ACHONDROPLASIA IN THE PIGEON

W. F. HOLLANDER

Carnegie Institution of Washington, Cold Spring Harbor, N. Y.

NORMAL AND ACHONDROPLASTIC PIGEONS

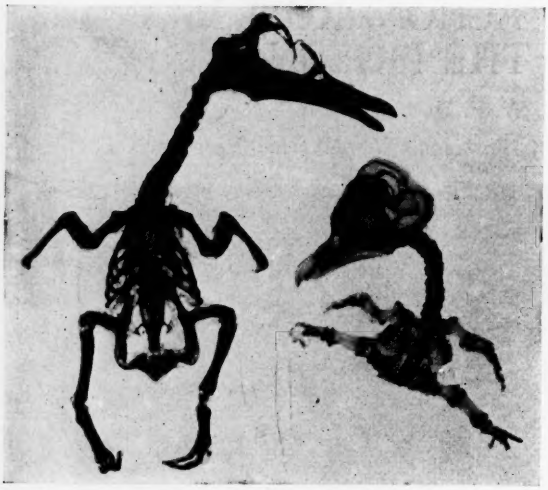
Figure 4

Three embryos unable to hatch. Left: a normally proportioned embryo; center and right, achondroplastic embryos, male and female, respectively, from mating 227. Failure to draw in the yolk sac is a general symptom of embryonic weakness. (Down of the achondroplastic embryos is normal while the other embryo is genetically short-downed.)

IN 1939 the writer purchased several pairs of White King pigeons from a breeder in Austin, Texas. One of the pairs, mating 20, produced within the following year six embryos that died at the hatching stage and were found to be uniquely abnormal. None succeeded in breaking the eggshell; in fact, there was no indication that even an attempt was made to break the shell. All these embryos had very short limbs and beak, about half normal length, like those illustrated in Figure 4. Dissection revealed that both sexes were represented, and no anomalies of the internal organs were seen. The thyroid glands appeared normal. Since the skeletal system seemed most affected, the term achondroplasia is used to designate the condition. The extraordinary shortening of all the bones, except perhaps the cranium, is shown by the alizarin stain technique (Figure 5).

It seemed probable that the achondroplastic type was a hereditary lethal, rather than an effect of nutritional deficiency or other environmental influence. The pedigrees of the parent pigeons, ♂ *WKA* and ♀ *WKB*, were not available, but it was known that they were close relatives. Even more informative is the fact that with each of the six achondroplastic embryos was a perfectly normal clutch-mate. Since no true lethals have previously been reported in the pigeon, except for a note by Lienhart[3] on a possibly sex-linked lethal, a further study seemed desirable.

Two offspring, both male, were raised to maturity from mating 20. One of these sons was next mated with his mother, and the father was meanwhile



SKELETON MUCH SHORTENED

Figure 5

Embryos cleared and stained with alizarin. Left: normal, eyes removed; right, achondroplastic, eyes not removed. Slightly enlarged.

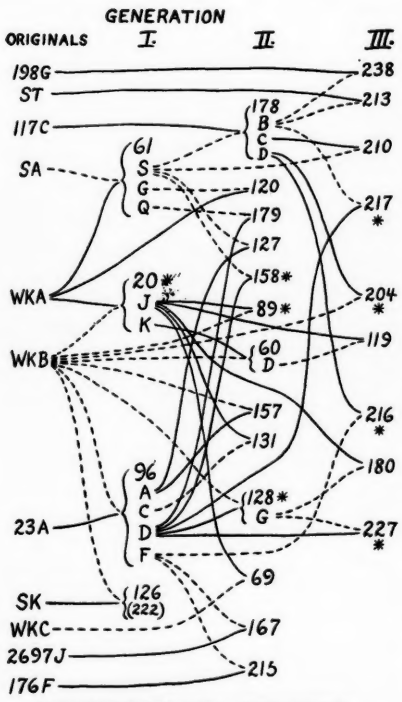
out-crossed to an unrelated female, *SA*, half Racing Homer and half King. Both of these new matings produced only normal offspring, some of which were allowed to mature. After producing a dozen young with her first son, *WKB* was mated with the other son, and soon produced a typical achondroplastic embryo.

The subsequent breeding program has been two-fold: production of breeding stock from out-crosses of achondroplasia producers, and progeny-testing of the new stock birds in matings with achondroplasia producers. A pedigree network of all matings is given in Figure 6).

Progeny tests were generally discontinued as soon as an achondroplastic embryo appeared, or else after eight or more normal offspring had been produced. Loss of the birds being tested prevented complete tests for matings 119 and 120. The results of the breeding to date are summarized in Table I. Of the 14 progeny-tested descendants of the original male and female, seven actually produced achondroplastic embryos. Such a proportion is exactly what would be expected if the achondroplasia depended on a single recessive factor; if two or more mutant factors were involved, a smaller proportion of out-cross birds would be capable of producing the type.

No achondroplastic embryos were produced in any out-cross. This fact indicates that no chromosome aberration, such as a translocation, is involved. Sex linkage is also ruled out. And since the ratio of achondroplastic : normal offspring in matings that produced achondroplasia is a satisfactory approximation to 1:3, it seems clear that a recessive autosomal mutant factor is responsible. The factor symbol *ac* is proposed.

Comment seems necessary on two other items in the table. First, mating 127 produced an offspring marked questionably achondroplastic. There are two reasons for believing that this was really normal: its limbs and beak were only moderately shortened and it managed to break the shell and nearly hatch before dying; and the father of this specimen produced no other abnormal young either in this test mating or another, 157. However, the dubious character of this embryo seemed worth calling attention to, especially in view of the uniformity of all the other achondroplastic specimens. The second item is the high proportion of eggs and embryos giving no



ACHONDROPLASIA PEDIGREE

Figure 6

Pedigree network including all matings and breeding birds involved in the study of achondroplasia. Numbers refer to matings (= sibships), letters to individuals. An asterisk indicates that achondroplasia was produced. Dash lines are from dams, solid lines from sires.

record among the matings which produced achondroplasia. Such losses were significantly lower in the out-crosses. One reason may be the rather large size of the birds in the former group—there was more likelihood of egg breakage. It may be a matter of heterosis however, at least in embryo survival to the hatching stage.

In addition to the factor (*s*) for white plumage, the original White Kings proved to be heterozygous for the "spreading" color factor, *S*. No indication of linkage between achondroplasia and *S* or white (as indicated by beak color in the embryo) has been found.

Observation of living achondroplastic embryos has been possible in several cases. If one egg of a clutch has hatched, and the other shows by candling that the embryo is alive but has made no effort to break the shell, one can be fairly certain the latter is abnormal. It has proved impossible to "rescue" these embryos; the rich blood-vessel supply of the embryonic membranes hemorrhages fatally when injured, and the embryos are too weak to start breathing.

The lethal type reported here is superficially similar to the chondrodystrophic and micromelic types, hereditary[1,2] and induced,[4] known in chickens. The "parrot beak" so generally known in such fowl embryos is not a characteristic of the achondroplastic pigeon embryos, however, and the bone defect seems somewhat different.

It is a pleasure to acknowledge the cooperation of Miss Elsie N. Ward, who prepared the alizarin-stained embryos.

TABLE I. Summary of breeding from all matings.

MATING #	TOTAL EGGS	EGGS LOST BROKEN OR INFERTILE	EMBRYOS DEAD, NO RECORD	RECORDED OFFSPRING	
				NORMAL	ACHONDROPLASTIC
ACHONDROPLASIA PRODUCER × ACHONDROPLASIA PRODUCER					
20	18	—	4	8	6
89	2	—	—	1	1
128	12	1	6	4	1
158	8	4	—	2	2
180	20	15	2	3	—
204	6	—	—	5	1
216	12	3	1	7	1
217	2	—	—	1	1
227	14	5	2	5	2
TOTAL	94	28	15	36	15
ACHONDROPLASIA PRODUCER × RELATIVE (PURE NORMAL?)					
60	12	—	—	12	—
119	5	2	1	2	—
120	2	—	—	2	—
127	6	—	1	4	(1?)
131	20	9	3	8	—
157	14	—	1	13	—
179	16	3	1	12	—
210	12	—	3	9	—
TOTAL	87	14	10	62	(1?)
ACHONDROPLASIA PRODUCER × UNRELATED PIGEON					
61	18	2	1	15	—
69	6	2	—	4	—
96	6	—	—	6	—
126(222)	9	1	—	8	—
167	12	2	—	10	—
178	4	—	—	4	—
213	8	2	—	6	—
215	2	1	—	1	—
238	4	—	—	4	—
TOTAL	69	10	1	58	0

Literature Cited

1. Asmundson, V. S. *Jour. Hered.* 33:328-330. 1942.
2. Lamoreux, W. F. *Jour. Hered.* 33:275-283. 1942.
3. Lienhart, R. *Comptes Rendus Soc. Biol.* (Paris) 126:336-342. 1937.
4. Lyons, M., and W. M. Insko. *Kentucky Agric. Exp. Sta. Bul.* No. 371.

◆

Research in Biology and Nuclear Physics

Chicago, Sept. 20, 1945.—As a further outgrowth of its work on the atomic bomb the University of Chicago has established an institute for applying the results of nuclear physics research to such problems as cancer, heredity, and the aging process. The institute, to be known as the Institute of Radiobiology and Biophysics, also will seek to perfect techniques for the protection of workers using radioactive materials. This need has been emphasized by the work on the atomic bomb and by the prospect of an increasing use of such materials in industry, Chancellor Hutchins said. The institute, to be a part of the division of biological sciences at the university, will be headed by Professor Raymond E. Zirkle, a botanist who has specialized in the effect of radiations on living organisms.

Members of the new institute will work in close cooperation with those of the Institute of Nuclear Studies already founded at the University of Chicago under the direction of Professor Samuel K. Allison, physicist. The latter institute has brought to the university faculty two additional Nobel Prize scientists, Enrico Fermi, the physicist, and Harold Urey, the chemist.

Establishment of the new institute is part of the university's plan to further fundamental research in fields which went underground or were neglected during the war. In commenting on Chancellor Hutchins' announcement, Dr. Reuben G. Gustavson, eminent biochemist and vice-president of the university, pointed out some of the promises and dangers of nuclear radiation:

"The studies in nuclear physics have revealed many types of radiation, some of which can be produced by new methods which allow accurate control. These new tools are keys with which to open the secrets of our physical and biological worlds. These tools are dangerous to work with, and man has to learn how to protect himself while using them.

"The application of these radiations to living tissues, organisms and biological systems is in its infancy. We can make new attacks on disease. For example, science now knows some two hundred chemical compounds which cause cancer. By replacing the normal atoms in these compounds with radioactive atoms of the same elements, these compounds can be followed in the animal body by a tracer method, and the methods by which they produce cancer may possibly be discovered."

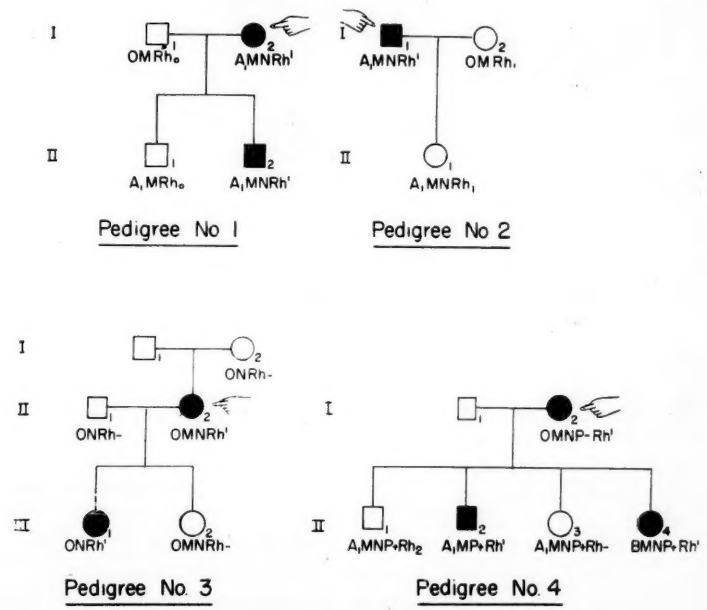
The action of drugs also can be followed by this method, Dr. Gustavson said. Common elements made radioactive by new methods can be incorporated into hormones, vitamins, and foods also, he added. Such problems as just how sugars and fats are burned within the body may be solved for the first time by this "tracer" method, he said.

Using these methods to probe deeper into the structure and activities of the living cell, scientists may also throw new light on heredity in plants and animals and the processes by which living organisms grow old, Dr. Gustavson said.

HEREDITY OF THE Rh BLOOD TYPES*

III Observations on the Rare Genes *Rh'* and *Rh''*

EVE B. SONN AND ALEXANDER S. WIENER
Serological Laboratory of Office of the Chief Medical Examiner of New York City



FAMILIES ILLUSTRATING TRANSMISSION OF GENE Rh'

Figure 7

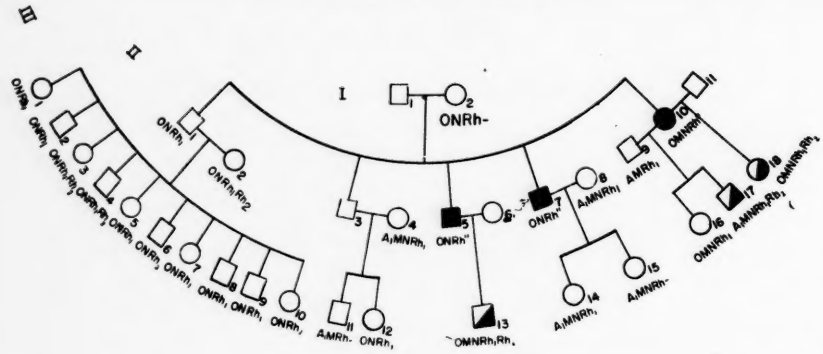
Rh' is very rare and these three pedigrees furnish additional data confirming the earlier conclusion that *Rh'* is an allele dominant to the Rh-negative allele (*rh*).

THE Rh types are of interest to the geneticist because they furnish a striking example of Mendelian inheritance in man.

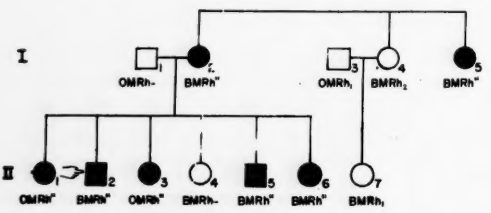
From patients who have had hemolytic reactions caused by the Rh factor, and from mothers of erythroblastotic babies, it has been possible to obtain sera containing three different varieties of anti-Rh agglutinins.[1,2,5,6] Using these reagents in agglutination tests, eight types of blood can be distinguished[3,4] as is shown in Table I. Wiener[3] has postulated that these Rh types are transmitted by six allelic genes, designated to correspond with the agglutinogens which they determine, as shown in Table II. These alleles pair to form 21 genotypes, and it is not difficult to ascertain which genotypes correspond to each of the eight Rh types (cf. Table III).

In previous papers [2,7,8] data were presented on the distribution of the Rh types in the population, as well as in a series with 97 families with 275 children. These results were in conformity

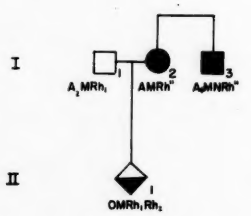
*Aided by a grant from the Carnegie Foundation through the Committee of Human Heredity of the National Research Council.



Pedigree No 5



Pedigree No 6



Pedigree No 7

THREE PEDIGREES OF THE INHERITANCE OF Rh"

Figure 8

Rh" is even rarer than Rh'. The data in these three pedigrees confirm the earlier conclusion that *Rh"* is another allele dominant to the Rh-negative allele (*rh*).

with the theory of multiple alleles, but the data were inadequate to test the theory in relation to the rare genes, *Rh'* and *Rh"*. The purpose of the present report is to present a number of pedigrees illustrating the transmission of *Rh'* and *Rh"*.

The pedigrees were compiled in the course of our studies in the distribution of the Rh types in the general population. Whenever an individual was encountered who belonged to either of the types Rh' or Rh", efforts were made to test other individuals in the family.

In Figure 7 are presented three pedigrees illustrating the transmission of gene *Rh'*. In general, type Rh' individuals may be assumed to be of genotype *Rh'rh,* because homozygous individuals (genotype *Rh'Rh'*) are very rare. In Pedigree 1, the father, who is of type Rh_o, is evidently heterozygous (genotype $Rh_o rh$) because one of the children belongs to type Rh'. The two children in this family like the two parents, must both be heterozygous. Pedigree 2 is too small to permit any conclusions as to the genotypes of either of the parents of the single child. In Pedigree 3, involving a mating of a type Rh' individual with an Rh-negative individual, the expectancy is that half the children will type Rh' and half Rh negative. In fact, there was one child each of these types in the family. In Pedigree 4, the father was no longer alive, but his Rh type can be deduced from the types of the four children and the mother. The oc-

currence of Rh_2 and Rh negative in this pedigree proves that the father must have belonged to type Rh_2, genotype *Rh_2rh*. It can also be seen that he must have belonged to group A_1B, type M or type MN and was P positive.

In Figure 8 are given three pedigrees illustrating the transmission of gene *Rh″*. In Pedigree 5, the grandmother was Rh negative, three of her children belonged to type Rh″, one to type Rh_1, while one was not available for testing. The grandfather was dead, but it is obvious that his blood must have belonged to type Rh_1Rh_2, genotype *Rh_1 Rh″*. It is also apparent that the type Rh_1 son (II-1) belongs to genotype *Rh_1rh* and, inasmuch as his wife belongs to type Rh_1Rh_2, it is to be expected that half the children would belong to type Rh_1, one-fourth to type Rh_2 and one-fourth to type Rh_1Rh_2. Actually, among the 10 children in this family there were six of type Rh_1, and two each of types Rh_2 and Rh_1Rh_2, in excellent agreement with expectations. The single grandchild (III-13) which the son (II-5) had belongs to type Rh_1Rh_2 so that his genotype is evidently *Rh_1Rh″*, even though the type of the mother is not known. As indicated in the diagram, II-7 was the individual whose blood we first found to belong to Rh″. That his wife, of type Rh_1, belongs to genotype *Rh_1rh* follows from the fact that one of their children is Rh negative. In this mating one-fourth of the children would be expected to belong to each of the four types, Rh_1, Rh_1Rh_2, Rh″, and rh negative. Since there are only two children, it is not surprising that there were no Rh″ children. It is evident that, II-17 and II-18 both belong to genotype *Rh_1Rh″* because their mother belongs to type Rh″.

As is indicated in Figure 8, Pedigree 6 was investigated because individual II-2 had been found to belong to type Rh″. Since type Rh″ individuals can usually be assumed to belong to genotype *Rh″rh*, a mating of such an individual with an Rh-negative person would be expected to yield half type Rh″ and half Rh-negative children. Mating I-1 × I-2 in Pedigree 6 yielded five type Rh″ and one Rh-negative individual. Pedigree 7 was studied because I-2 was the mother of an erythroblastotic baby (II-1).* (It might be remarked that of the three factors, Rh_0, Rh′ and

TABLE I. Classification of the Rh blood types.

Bloods lacking the Rh_0 factor			
Reactions with antisera			
Anti-Rh′	Anti-Rh″	Anti-Rh_0	Types
−	−	−	Rh-neg.
+	−	−	Rh′
−	+	−	Rh″
+	+	−	Rh′Rh″
Bloods containing the Rh_0 factor			
Reactions with antisera			
Anti-Rh′	Anti-Rh″	Anti-Rh_0	Types
−	−	+	Rh_0
+	−	+	Rh_1 (Rh_β)
−	+	+	Rh_2 (Rh_β)
+	+	+	Rh_1Rh_2($Rh_\beta Rh_\beta$)

TABLE II. The 6 Rh genes and the reactions which they determine.

Rh genes	Reactions with antisera		
	Anti-Rh_0	Anti-Rh′	Anti-Rh″
rh	−	−	−
Rh′	−	+	−
Rh″	−	−	+
Rh_0	+	−	−
Rh_1	+	+	−
Rh_2	+	−	+

TABLE III. The 8 Rh types and their 21 genotypes.

Rh types	Genotypes
Neg.	rhrh
Rh′	Rh′Rh′ and Rh′rh
Rh″	Rh″Rh″ and Rh″rh
Rh′Rh″	Rh′Rh″
Rh_0	Rh_0Rh_0 and Rh_0rh
Rh_1	Rh_1Rh_1, Rh_1rh, Rh_1Rh', Rh_1Rh_0 and $Rh'Rh_0$
Rh_2	Rh_2Rh_2, Rh_2rh, Rh_2Rh'', Rh_2Rh_0 and $Rh''Rh_0$
Rh_1Rh_2	Rh_1Rh_2, Rh_1Rh'' and $Rh'Rh_2$

*For the blood samples from this pedigree we are indebted to Dr. Philip Rosenblatt.

Rh", Rh_0 is by far the most antigenic and therefore the most important clinically. For this reason, mothers of types Rh′ and Rh" are on almost the same level with rh-negative mothers with regard to the likelihood of their having erythroblastotic babies. While child II-1, in Pedigree 7 had two properties, Rh′ and Rh_0, lacking from his mother's blood, it is probable that property Rh_0 played the predominant role in the isoimmunization.)

Summary

Four pedigrees are presented illustrating the transmission of the rare gene *Rh′*, and three pedigrees illustrating the transmission of the rare gene *Rh"*. These data lend further support to Wiener's theory of inheritance of the Rh blood types by multiple allelic genes.

Literature Cited

1. Wiener, A. S. *Arch. Path.* 32:227, 1941.
2. ————. *Science* 98:182, 1943.
3. ————. *Proc. Soc. Exp. Biol. and Med.* 54:316,1943.
4. ————. *Science* 99:532, 1944.
5. Wiener, A. S., and Landsteiner, K. *Proc. Soc. Exp. Biol. and Med.* 53:167. 1943.
6. Wiener, A. S., and Sonn, E. B. *Jour. Immunol.* 47:461. 1943.
7. Wiener, A. S., Sonn, E. B., and Belkin, R. B. *Proc. Soc. Exp. Biol. and Med.* 54:238. 1943.
8. ————. *Jour. Exp. Med.* 79:235. 1944.

✦

GENETICS IN HOLLAND

UNDER date of September 7, a letter has been received from Professor Dr. M. J. Sirks, Director of the Genetisch Institute, Huis de Wolf, Haren (Gron.) Netherlands. It has news of several geneticists well known in America. He writes as follows:

"You ask for information about conditions in Europe. I can tell you that we try to repair with all our forces the immense damage the Germans have brought to our country. The Universities of Leyden, Amsterdam, and Utrecht have not been damaged much, because in these towns there was no fighting. Though the town of Groningen has suffered much by fighting, the buildings of the University (i.e. the Genetical Institute) have escaped serious damage. The buildings of the University College of Agriculture at Wageningen have been damaged heavily; all glass is destroyed (greenhouses entirely) and such cannot be repaired for a year or so, because of lack of materaials; doors, floors, and floor joists have been stolen and the conditions of the buildings are very bad. Instruments and archives have been stolen or burnt; a few things escaped the attention of the Germans, but on the whole the buildings have been looted of almost everything.

"About the geneticists themselves we may mention that Dorst, Hagedoorn, Honing, Sirks, Stomps, Miss Tammes and Wellensiek are still alive, but all have suffered either by forced evacuation, the continuous mental strain, caused by the German repression, or by the starvation campaign by which the Germans tried to press down the resistance in the Netherlands. The human geneticists Bijlmer, Frets and Waardenburg are still alive, but Sanders, who was a Jew has probably been brought to Germany, no news having been received since 1941. Waardenburg has lost his valuable library; he lived in the destroyed town of Arnhem. Measures by which the Germans tried to lay hands on all students with the intention to bring them like slaves to Germany have failed for the greater part by the resistance of the students, but resulted in closing the Universities in May 1943; since at that time no scientific work could be done, since no man between 17 and 45 dared to come into the streets because of the razzias. Assistants slept for some time in the laboratories, but as this also proved to be dangerous, they 'sank below the horizon' and disappeared into the unknown. The greater part of experiment gardens has been used for

growing vegetables. We had to do much other work, i.e. 'underground,' and we had to keep in the shadow because we did not like to be put in jail by the Germans.

"Now we are resuming our activities: the Universities have opened already or will open very soon; assistants and students have come back, if not murdered by the Germans or died in a concentration camp. We are also resuming our relations with friends and colleagues in other countries; though postal connections are still very slow, we are very glad to receive more and more foreign letters. I can tell you that in Denmark Winge, Jorgensen and Skovsted are still alive; that Mohr (Oslo) has been in a German concentration camp, where he was very ill, but he is still alive. Federley (Helsinki) is all right. I assume that from Great Britain you will have received enough information. From Germany I only know that F. V. Wettstein died in February 1945 by inflammation of the lungs, while Renner has been seriously wounded. Since May 1940 however I had no correspondence with German colleagues or friends; I only received in a secret way a letter from Mrs. Wettstein informing me about the death of her husband.

"One of the things we now want most is scientific literature, books, periodicals, and reprints. You would do me a very great pleasure by asking the American geneticists to send everything they can spare, published since the spring of 1939 on genetics (including cytology, sex, human genetics, and animal and plant breeding). We have a lack of knowledge of about six years and that also should be repaired. The address of my Institute is: Genetisch Instituut, Huis de Wolf, HAREN near Groningen, Netherlands."

◆

GENETICS AND CLINICAL PROBLEMS

THE adaptation of genetics to clinical problems and the development of the medico-genetic viewpoint have been the subject of several recent texts. The second edition of this volume* by a physician well versed in genetics is one of the best to appear. It will appeal to the clinician because of its practical medical approach, written in language that the practising physician will understand and appreciate. It is also of prime importance to the geneticist in revealing to him the very practical importance of his subject in human health and disease, and the very great strides that human genetics has made in the last decade or two.

Although the book is in no sense a genetics text, it provides, in connection with the discussion of specific anomalies, an introduction to the simpler genetic mechanisms. Even the more abstruse topics are touched upon. The concept of linkage is brought up several times, as in the hereditary complexes associated with the Laurence-Moon-Bardet-Biedl syndrome, and the syndrome of fragility of bones, blue sclerae and hardness of hearing. Lethals are discussed, including not only the common recessive lethals, but the rarer semi-dominant lethals such as Pelger's anomaly.

The author stresses the fact, now well known to students of human genetics, that different genes may produce indistinguishable phenotypic effects, while on the other hand single genes may extend their activities to several quite different parts and functions of the body. In this connection he discusses his views of pleiotropism and its application to such conditions as epiloia, phenylpyruvic oligophrenia, and the association of physical type with temperament.

The author states that "For those, however, who see human genetics merely as deciding whether a particular anomaly is transmitted as a dominant,

*_Constitution and Disease_, by JULIUS BAUER, M.D. xiii + 247 pp. New York, Grune and Stratton, 1945.

recessive, or sex-linked characteristic, or whether it is due to one or more abnormal genes, it might be well to emphasize with Fraser Roberts that it is impossible to settle once and for all the mode of a trait and thereafter to apply that knowledge to each new case that presents itself."

The Degenerative Status

Much is made by the author of a condition, or combination of conditions, called by him "status degenerativus." In defining normality and abnormality, he considers a variation in a trait abnormal if it is found in less than 4.5 per cent of the population. An accumulation of such abnormalities in a person brings about, according to the author, a state of biologic inferiority, lower resistance, greater morbid predisposition, resulting in a diminished adjustment to the environment. The geneticist will think twice before accepting "status degenerativus" as a significant classification of constitution, although the author's contention that it behooves the physician to take into consideration not one or several organs of the patient, but his whole personality, both physical and mental, is certainly sound.

An amazing number of anomalies, diatheses and diseases is discussed in the course of the volume. Citations are frequent and complete, more than 400 references being listed, most of them very recent. One cannot help being impressed with the author's wide experience, his thorough familiarity with both domestic and foreign literature, his sincere appreciation of the genetic viewpoint, and his ability to make his views clear and interesting to the reader. A good index and several clear figures accompany the text.

LAURENCE H. SNYDER
Ohio State University, Columbus 10

✦

POPULATION PROBLEMS

THE authors maintain that "the task of freeing all of the people of the world from war and want and fear is chiefly the job of establishing a favorable relationship between number of people, on the one hand, and on the other hand, the natural resources and technical developments, in all the countries of the world."* Much of the world is already over-populated, yet in times of peace the world population was growing at a rate which would double the number of people in only 70 years. Even if the net reproductive rate could be reduced to 10 per cent below replacement levels it is estimated that the world population would reach a total of 2,750,000,000 before it would begin to decline.

It is difficult for us to realize that three-fourths of the people of the world are living at little more than subsistence levels, with real incomes per family of less than ten dollars per week. These people have high birth rates and population growth is held in check by high death rates, yet their numbers increase as fast as resources and public health permit.

In the more prosperous countries of the world the birth rates have declined, and in many nations are below replacement levels. From the standpoint of per capita welfare most European countries and even the United States, are better off with smaller populations. Much of our own prosperity is due to the utilization of our capital reserves of natural resources.

The authors show that population pressure is one of the major causes of war. Hitler and Mussolini appealed for higher birth rates while at the same time demanding more space for their peoples. The politcal leaders of both England and France are now appealing

*POPULATION ROADS TO PEACE OR WAR, by GUY IRVING BURCH and ELMER PENDELL. Population Reference Bureau, Washington, D. C. 1945.

for higher birth rates in these overpopulated countries. Larger populations can lead only to lower living standards and may adversely affect military security. Modern war demands a relatively high standard of living to provide the surpluses of material and production resources. The excessive populations of China and India are a military handicap because they produce so little in excess of the goods needed for mere subsistence.

The discussion of differential birth rates among the various economic and educational groups in the United States emphasizes the undesirability of the excessive fertility of the poor and the ignorant. In the words of John Stuart Mill "Little advance can be expected in morality until the producing of large families is regarded with the same feeling as drunkenness or any other physical excess."

Although the authors show that migration can not reduce population pressure in Asia, they do not stress the great difficulties in adjusting populations to resources in these densely populated countries. Even in the modern Western nations the phenomenon of differential fertility presents problems of complex relationships which can be solved only with great difficulty. Yet these problems must be solved if all men of all nations are to be freed from war and want and fear. As Pitkin points out in the introduction, they cannot be solved through a return to some ancient moral code.

KARL SAX

Harvard University

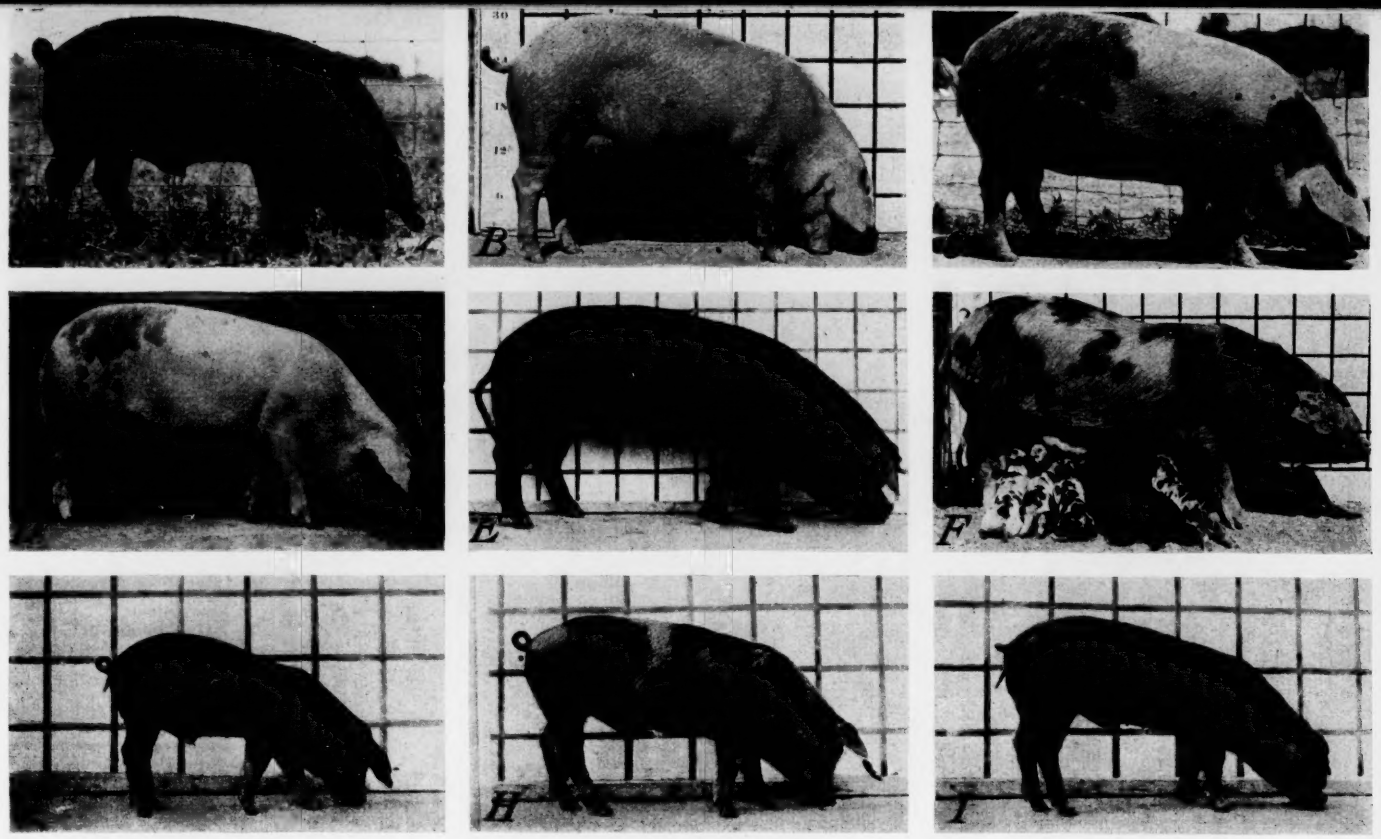
✦

An Abstracting Service for Human Biology

The Trustees of *Biological Abstracts* announce the establishment, beginning in January, 1946, of a new section of *Biological Abstracts* — Section H, specially assembled Abstracts of Human Biology — intended for anthropologists, sociologists, psychologists, neurologists and psychiatrists, students of child development and human welfare, and students of man generally.

The new section will be an assemblage of all abstracts published in *Biological Abstracts* dealing with the broad field of human and social biology. Biological studies on human inheritance, on population and fertility, on endocrine and neurological factors affecting growth, development and human personality, on alcoholism and drug addiction, and on nervous disorders and mental deficiencies, and broad nutritional and epidemiological studies affecting human welfare, are some of the many fields that will be covered. The annual subscription price for the ten abstract issues, plus the complete index of the year's volume of *Biological Abstracts* will be $6.00 ($6.50 outside the United States).

Full information may be obtained by writing to Mr. H. I. Anderson, Business Manager, Biological Abstracts, University of Pennsylvania, Philadelphia 4, Pennsylvania.



HYBRIDS OF LANDRACE AND LARGE BLACK PIGS

Figure 9

A—Boar 41, one of the two Large Black boars imported by the U. S. Department of Agriculture from England in 1936. This boar sired 75 of the 90 F_1 pigs in matings with six Landrace sows. One of the sows, sow 11.11, is shown in *B*. *C*—F_1 gilt 9.1 out of sow 11.11 by boar 41. The size and distribution of skin spots on this gilt are typical of most F_1 pigs. *D*—Topcross gilt out of Landrace sow by a black backcross generation boar, showing slightly less than the average amount of skin spotting on topcross pigs. *E*—Boar 1874 out of a solid black F_2-equivalent sow by a solid black F_2-equivalent boar. This boar, himself a solid black, when mated to two black-spotted sows of the Landrace × Poland China cross produced 8 black and 19 black-spotted pigs. One of the litters which were sired by boar 1874 is shown in *F*. Note that the black spotted pigs closely resembled their dam as regards amounts of black present. All but one of the five black pigs produced by this sow had a little white on their feet. *G*—A black pig with as much white on the feet as was observed among pigs known to carry the gene *E*. Pigs shown in *H* and *I* were produced by $E\ E^p \times E\ E^p$ type of matings. These pigs, showing a sandy and red ground color, had about the average amount of black on black-spotted pigs.

INHERITANCE OF COAT COLOR IN SWINE

IV. Analysis of Hybrids of Landrace and Large Black*

H. O. HETZER†

United States Department of Agriculture

EARLIER papers[2,3] by the author have reported an analysis of the color factors differentiating the Poland China and Berkshire from the Landrace. It was shown that the black color of the Poland China and of the Berkshire behaves as a simple recessive to the white of the Landrace. It was also shown that the Poland China and the Berkshire have minor factors for extension of black, while the Landrace has minor factors which tend to restrict black. These findings, together with results of a cross between a Landrace × Poland China and Landrace × Berkshire hybrid, discussed in one of these papers[3] furnish definite proof that the black color of the Poland China depends on the same major gene as that of the Berkshire. Physiologically, the main facts were explained on the hypothesis that the Landrace carries the same black-spotting gene E^p‡ as do the Poland China and the Berkshire, and with it a dominant gene for white, *I*, which is epistatic to black. On this hypothesis the Landrace would have the formula $E^p\ E^p\ I\ I$, and the Poland China and the Berkshire $E^p\ E^p\ i\ i$.

The purpose of the present paper is to present the evidence supporting the above mentioned hypothesis and to demonstrate that the solid black color of the Large Black depends on a dominant allele of the black-spotting gene E^p possessed by the Landrace. The same symbols have been used as in earlier reports, with the addition of a new symbol *E*, which represents the gene for complete extension of black as seen in the Large Black.

Three of the five Large Blacks used in the experiment belonged to a group of two boars and two sows imported by the United States Department of Agriculture from England in 1936, while the other two traced to those that were imported. Unlike the Poland China or Berkshire, all the Large Blacks have been solid black without any white markings. The 36 Landrace animals used in the study came from the same source as those used in the crosses with the Poland China and the Berkshire. They have been white or white with a few skin spots.

The Hybrids

The experiment was begun by mating two Large Black boars to eight Landrace sows. The 90 F_1 pigs from these matings were white or white with varying numbers of black skin spots on the back, neck, face, and ears (Figure 9). In most pigs, the hairs on the spots were white, but some spots had a distinct bluish appearance due to an intermixture of black and white hairs. Some of the F_1 pigs also had longitudinal stripes. The pigs showing black color in the skin had larger spots, on the whole, than the F_1's from the Landrace × Poland China and Landrace × Berkshire crosses described previously. No attempt will be made to explain further the incidence of skin spotting, except to suggest that the Large Black has a more effective factor complex for density of black than the Poland China or the Berkshire.

*Data obtained at the Beltsville Research Center, Beltsville, Md.

†The author acknowledges his indebtedness to Mr. J. X. King for recording the colors of the pigs used in this study.

‡In the two preceding papers of this series the symbol E^p was erroneously printed E^p. Since E^p is an allele of *E*, the symbol E^p will be used hereafter to designate the spotting allele.

Eight sows and two boars were selected from the F_1 generation for breeding purposes. Seven of the F_1 sows when mated with the two F_1 boars produced a total of 84 F_2 pigs. Of the 84 pigs, 57 were white, 21 black and six black-spotted (Table I). This is in good agreement with a 12:3:1 modified dihybrid ratio. The conclusions indicated by these findings are that the white of the Landrace depends on a single dominant gene (*I*) epistatic to both solid black (*E*) and black spotting (E^p), that solid black is dominant over black spotting and that the two genes are inherited independently of each other. Accordingly we have no hesitation in assigning the formula $E^p\ E^p\ I\ I$ to the Landrace, $E\ E\ i\ i$ to the Large Black and $E^p\ E^p\ i\ i$ to black-spotted animals.

Five of the F_1 sows when backcrossed to two Large Black boars produced 53 pigs, while one of the F_1 boars when backcrossed to two Large Black sows produced 18 pigs. (See Table I.) In both cases equal numbers of white and black pigs were expected. The results from the backcross to Large Black sows are in perfect agreement with this expectancy, but there is a considerable deficiency of whites among the pigs from the reciprocal cross (exhibited equally by sons and daughters). The ratio here is 18 white to 35 black, a deviation yielding a value for *P* of less than 0.02 by the χ^2 test. Obviously this deficiency cannot be attributed to linkage between the above mentioned genes, for even if they were linked the frequencies of the two classes should conform to a 1:1 ratio. Before further interpretation of these data is attempted it will be well to examine the results from other crosses made during later stages of the experiment.

Results in Later Generations

Following the general plan of the experiment as previously described,[1] a total of 1,670 pigs was produced in addition to the F_1, F_2, and backcross generations. Of these 1,670 pigs, 507 were produced by crossing black segregates of the F_2 and backcross generations (as well as of other segregating generations) back to the Landrace (topcross pigs), 437 were produced by intercrossing topcross animals (F_2-equivalent pigs), 408 were produced by backcrossing black segregates to topcross animals (backcross-equivalent pigs), and 318 were produced by intercrossing black segregates. A summary of the four progeny groups is presented in the lower part of Table I.

The 507 topcross pigs closely resembled the F_1 pigs, indicating that their Landrace parents (19 Landrace boars mated with 22 black sows and three black boars mated with nine Landrace sows) all carried the inhibitor *I* in a homozygous condition.

The classes of young recorded among the 437 F_2-equivalent pigs, except for a few minor points to be discussed later, were the same as those obtained in the F_2 generation. Disregarding the classification into black and black-spotted, there are 340 white to 97 black-carrying pigs, in good agreement with the expected 3:1 ratio, proving conclusively that white is due to a dominant gene. The ratio of black to black-spotted pigs, 63:34, is more nearly a 2:1 than a 3:1 ratio. However, this is not surprising when we consider that many of the black segregates that were backcrossed to Landrace were heterozygous for black and that their offspring should consist of $EE^p\ Ii$ and $E^p\ E^p\ Ii$ individuals. Unfortunately only a few of the animals thus produced could be assigned to one or the other of the two categories. It is clear, accordingly, that these data can throw no light on the hypothesis that the inhibitor *I* and the black-spotting gene E^p, are inherited independently of each other.

The backcross-equivalent data in Table I show that black-spotted pigs may be obtained by matings of black with white animals. This readily explains the apparent excess of black-spotted pigs in the F_2-equivalent group, as many of the heterozygous blacks which were mated with topcross animals also had offspring in matings with Landrace. Combining the black and black-

spotted pigs gives 87 white to 65 black-carrying pigs from the (L × black) ♂ × black ♀ matings and 108 white to 148 black-carrying pigs from the black ♂ × (L × black) ♀ matings. While in both cases a 1:1 ratio would be expected, only in the case of the matings involving black sows are the deviations from this expectancy within limits reasonably attributable to random sampling (see Table I). The progenies from the matings involving white sows, on the other hand, show a considerable deficiency of the white class, a result very similar to that obtained by backcrossing F_1 sows to Large Black boars. These deviations are in the direction expected if some maternal influence were affecting color of offspring. In both cases the matings involving heterozygous white sows were the ones which gave the aberrant results. Such influence, if effective, should tend to make litters by black boars consist wholly of black-carrying pigs and litters by heterozygous white boars of equal numbers of white and black-carrying pigs. The fact that the F_2 and F_2-equivalent data reveal no such tendency practically eliminates this hypothesis.

Additional evidence against this hypothesis is furnished by the fact that only one or two F_1 sows (out of a total of five) and two topcross sows (out of a total of 13) were found which gave an indication of a real deficiency in the white class. The pigs out of the two F_1 sows consisted of six whites and 19 blacks (5:13, 1:6), while those out of the two topcross sows consisted of 12 whites and 46 black-carriers (5:16, 7:30). If these pigs are omitted, the ratios of the remainder, 12:16 instead of 18:35, and 96:102, instead of 108:148, do not differ materially from the theoretical 1:1 ratio. A point of considerable importance in this connection is that the pigs out of the four sows just mentioned were by five sires, and that all of these boars had about equal numbers of white and black (or black-spotted) pigs from matings involving other heterozygous white sows (9:13, 3:3, 20:27, 7:8, 21:17).

These results, when considered in conjunction with the fact that the ratio of whites to blacks among the progenies of the four sows is fairly close to a 1:3 ratio, can most simply be explained on the hypothesis that white depends on two dominant genes rather than on one, and that the four sows were heterozygous for both of them. In order to reconcile the hypothesis with the main body of data, the only assumption necessary is that some of the Large Blacks were heterozygous for the second dominant gene. The real test of the hypothesis lies in the possibility of producing whites from matings between blacks. Until the production of whites by such matings has been demonstrated, the above interpretation remains only tentative. The only other hypothesis which the author is able to offer in this connection is that the departures from the expected 1:1 ratio exhibited by the two sets of progenies represent extreme chance deviations.

The occurrence of black-spotted pigs among the progenies of the black × black matings in Table I affords definite proof that solid black is dominant over black spotting. Since some of the animals used in these matings were expected to be homozygous for black, the first step in testing the hypothesis that solid black depends on one major dominant gene was to separate all matings which produced both black and black-spotted pigs from those which produced only blacks. When this was done, it was found that 185 of the pigs in Table I were the product of matings between heterozygous animals while the remainder, 133 pigs, were produced by animals presumably homozygous for black. The 133 pigs from the latter type matings were all solid black, while the 185 pigs from the matings between heterozygotes consisted of 126 blacks and 59 black-spotted, where 138.8:46.2 were expected if a single gene is involved. There is a considerable excess of the black-spotted class, a result which suggests that the matings, which failed to produce black-spotted pigs included some in which both parents were heterozygous. The fact that the number of pigs produced

by these latter matings averaged only about eight pigs per mating supports this suggestion. It is most probable, therefore, that the failure of some of these matings to produce black-spotted pigs was merely a matter of chance. Accordingly, it seems safe to conclude that solid black differs from black spotting by one main dominant gene.

Appearance of Black and Black-Spotted Segregates

While all the pigs that were classified as black in the F_2 and backcross generations were solid black, many of those obtained in subsequent generations had a few white markings. Usually, only the front feet were involved, but occasionally all feet were white and a few pigs had white on the nose or tip of the tail (Figure 9). Whatever the mode of inheritance of these markings, there is evidence that solid black animals may transmit the white-footed condition, while white-footed animals may have solid black offspring. There is also evidence that the presence of white feet is not necessarily a sign of heterozygosis as regards the fundamental gene for black. Accordingly, it appears probable that the appearance of white feet is due to minor genes for restriction of black, presumably brought into the cross by the Landrace.

The main point of interest in regard to the pigs described as black-spotted is that they showed the same sort of spotting as was observed among the black-spotted Landrace × Poland China and Landrace × Berkshire pigs (Figure 9). This obviously suggests that the Large Black has similar genes for extension of black as does the Poland China or the Berkshire. Like the black-spotted Landrace × Poland China hybrids, the black-spotted pigs in this experiment had for the most part a white ground color, but some had a sandy and, less frequently, a red ground color. The appearance of sandy and red in combination with black is compatible with the assumption that the Large Black has certain red intensity factors, lacking in whole or in part in the Landrace. Because of the small number of pigs having sandy or red color, no attempt is made here to explain further these variations in ground color.

Summary

Crosses between the Landrace and the Large Black breeds have shown that the Landrace carries a major dominant gene for white (I) which is epistatic to the gene for self black (E) possessed by the Large Black. The Landrace also carries a gene for black spotting (E^p) which is recessive to the gene for self black (E). Both E and E^p are hypostatic to I. There are probably minor genes which either extend or restrict the effects of both the self-black and the black-spotting genes.

Gene I evidently is inherited independently of gene E^p, but additional data are needed before final conclusion can be drawn on this point.

The appearance of sandy-and-black and red-and-black spotted pigs in addition to white-and-black spotted pigs in some of the segregating generations is explained on the assumption that the Large Black has red intensity factors, lacking in whole or in part in the Landrace.

Literature Cited

1. Hetzer, H. O. *Jour. Hered.* 36:121-128. 1945.
2. ———. *Jour. Hered.* 36:186-192. 1945.
3. ———. *Jour. Hered.* 36:254-256. 1945.

Table I. Color of pigs in the F_2 and backcross generations of the Landrace x Large Black cross. Included are the results of various other crosses of the same original cross.

Name of crosses	Number and color of pigs				χ^2	P
	Total	White	Black	Black-spotted		
F_2 generation						
F_1 white x F_1-white	84	57	21	6		
Expected (12:3:1)		63.0	15.8	5.2	2.41	0.30
Backcross generation						
F_1-white ♂ x Large Black ♀	18	9	9	-		
Expected (1:1)		9	9	-	0.00	1.0
Large Black ♂ x F_1-white ♀	53	18	35	-		
Expected (1:1)		26.5	26.5	-	5.45	< 0.02
Reciprocal generation						
Landrace x Black	507	507	-	-		
Expected (all white)		507	-	-	-	-
F_2-equivalent generation						
(L x black)-white x (L x black)-white	437	340	63	34		
Expected (3:1)		327.8	109.2		1.83	0.18
Backcross-equivalent generation						
(L x black)-white ♂ x black ♀	152	87	47	18		
Expected (1:1)		76.0	76.0		3.18	0.08
Black ♂ x (L x black)-white ♀	256	108	118	30		
Expected (1:1)		128.0	128.0		6.25	< 0.02
Black x Black	-	318	-	259	59	-	-

TWO NEW PEDIGREES OF ZYGODACTYLY

With Notes on Its Partial Sex-Linkage

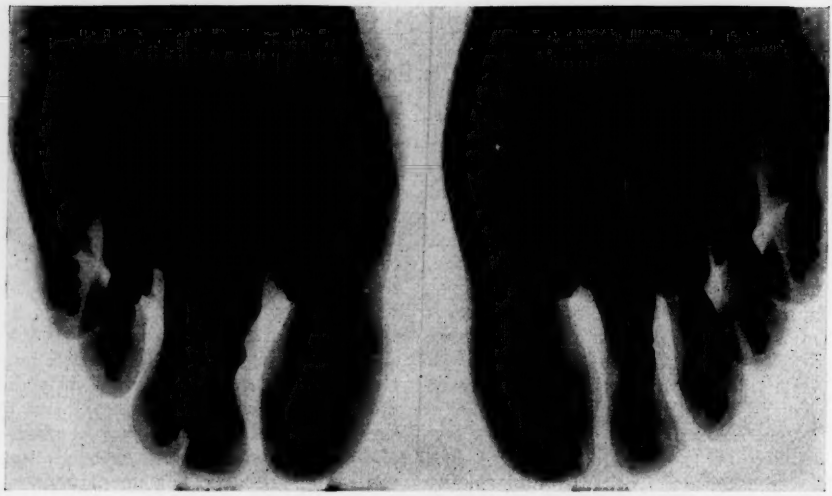
ALAN C. PIPKIN* AND SARAH BEDICHEK PIPKIN
Austin, Texas

WEBBING of the second and third toes with no change in bone structure has been reported by Hurlin, Schofield, Wright, Schultz, Castle, and Heston. The term zygodactyly was proposed by Weidenreich (quoted by Schultz) to distinguish simple skin webbing from other forms of syndactlyly in which the bones may be involved. With the exception of Roskoschny's case (quoted by Schultz), skin webbing of the second and third toes is not associated with any abnormality of the hands. Webbing between the second and third toes represents the persistence of a normal embryological condition as several authors have pointed out. The purpose of the present paper is to describe two new pedigrees of zygodactyly involving second and third toes only.

A photograph and an x-ray photograph of the two feet of the propositus, III-3, of pedigree *A* are given in Figures 10 and 11*A*. The photograph shows that zygodactyly occurs in the right foot only of *A* III-3. Webbing extends up to the toenails, but both nails are fully formed. A calloused area on the lower part of the second toe of the right foot is not present to such a degree on the unwebbed second toe of the left foot. Figure 10 reveals normal bone structure in the right foot of *A* III-3, although the position of the phalanges of the second and third toes is modified by the skin web holding these toes together. The propositus, *A* III-3, states that his father, *A* II-1, possessed a similar webbing of the second and third toes of his left foot, the right foot being normal. In Sommer's case (quoted by Schultz), the second and third toes were webbed on the right side only in all members of the pedigree.

*Lieutenant, H (S), USNR.

In Pedigree *A* (Figure 12*A*) a webbed toed man, II-1, whose aunt, I-1, was also affected, transmitted webbed toes to his son, III-3, the propositus. I-3, the father of II-1, was apparently a carrier of the abnormal webbing gene, although he appeared normal. No kinship existed between II-1 and II-6, parents of the propositus. Zygodactyly did not occur in the maternal family line of the propositus. If this short pedigree alone were considered, webbed toes would appear to be dependent upon an autosomal irregular dominant gene. However, Schultz pointed out that males were more apt to inherit zygodactyly than females in the three German pedigrees he reviewed. Information derived from the Pipkins' pedigree *A* and from six pedigrees given by Hurlin, Wright, Pfitzner (quoted by Schultz), Wolff (quoted by Schultz), and Heston strongly suggest that zygodactyly is dependent upon a dominant partially sex-linked gene in these pedigrees. Haldane showed that in certain pedigrees retinitis pigmentosa was dependent upon a dominant gene located in that part of the *X* chromosome which is homologous with and undergoes crossing over with a portion of the *Y* chromosome. Haldane describes such genes as being "partially" or "incompletely" sex-linked. Thus in dominant retinitis pigmentosa pedigrees, an affected man who inherited the defect from his father has the abnormal gene on his *Y* chromosome and therefore tends to pass the defect more frequently to his sons (non-crossovers) than to his daughters (crossovers). If the affected man inherited the anomaly from his mother, then the retinitis pigmentosa gene must be on his *X* chromosome. In this case he transmits the defect more frequently to his daughters (non-crossovers) than to his sons



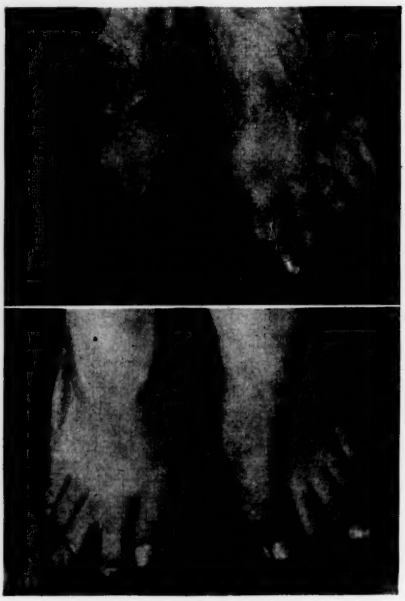
BONES NOT AFFECTED

Figure 10

X-ray photograph of the toes of propositus (*A* III-3). The position of the phalanges is modified by the close fusion of the toes of the right foot. The left foot has no web between the toes (see Figure 11*A*).

(crossovers). Affected individuals of like sex as the affected paternal grandparent and normal individuals of unlike sex as the affected paternal grandparent are non-crossovers. Conversely, affected individuals of unlike sex as the affected paternal grandparent and normal individuals of like sex as the affected paternal grandparent constitute crossovers.

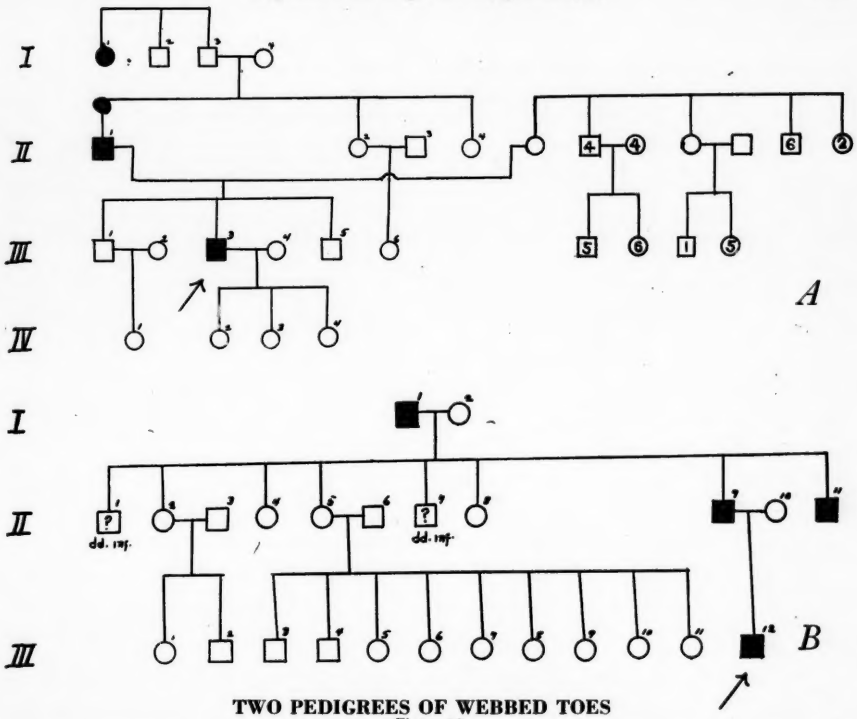
Applying Haldane's method to the seven zygodactyly pedigrees previously mentioned, Table I shows the total number of non-crossovers and crossovers and the percentage of crossing-over between zygodactyly and that part of the *X* chromosome not homologous with the *Y* chromosome. Critical marriages must involve an affected man and normal woman since if a woman is affected, crossing over between her two *X* chromosomes is not detectable. According to Table I, 15 crossover and 34 non-crossover progeny of 13 critical marriages occurred. The percentage of crossing over between zygodactyly and



ONE-SIDED WEBBING

Figure 11

A—Only the right foot of *A* III-3 shows webbing between the second and third toes and this is extreme (See also Figure 10). *B*—Usually both feet are affected and the webs are less extensive, as in the case of *B* III-12 of the pedigree *B*, Figure 12.



TWO PEDIGREES OF WEBBED TOES
Figure 12

Pedigrees of two families displaying webbing between the second and third toes. The inheritance could conform to a dominant partial sex-linked gene, with approximately thirty per cent crossing-over between the X and the Y chromosomes.

the differential segment is thus 30.6. The deviation from the 24.5 affected individuals of unlike sex plus normal individuals of like sex as the affected paternal grandparent expected in the absence of partial sex linkage is 9.5, with a standard deviation of 3.5. This deviation is well more than twice its standard deviation. Normal appearing carriers, who were detectable since they transmitted the trait, appear once in Heston's pedigree *A*, three times in Heston's pedigree *B*, once in Wright's pedigree, and once in the Pipkins' pedigree *A*. A few undetectable normal appearing carriers may have occurred, thus slightly lessening the accuracy of the computed crossover percentage. However, the authors feel that the evidence presented in Table I strongly suggests that zygodactyly in the cases mentioned is caused by a dominant partially sex-linked gene. The ratio of affected to normal progeny of all marriages with one parent affected in the seven pedigrees under consideration is 48:48. From webbed toed mothers, the ratio of affected sons to affected daughters is 9:5. Thus there is no evidence of sex limited modifiers. Haldane pointed out that an excess of affected daughters from affected mothers and an excess of affected sons from affected fathers is expected in the case of sex limited modifiers. Four pedigrees occur in the literature which were not used in the crossover calculations in Table I for reasons given as follows: Schofield's pedigree and the Pipkins' pedigree *B* show transmission in the male line only; i.e., no crossing-over takes place. In Castle's pedigree, females only are affected, so there are no critical marriages. Sommer's pedigree (quoted by Schultz) is

discarded because it contains 17 affected and only three normal persons in the entire pedigree. Even in this extraordinary pedigree the ratio of affected males to females is 11:6.

A photograph of the feet of the propositus, III-12, of pedigree *B* is given in Figure 11*B*. Webbing occurs between the second and third toes of both feet. The skin web extends more distally in the inter-digital space on the left foot than on the right, similar to Hurlin's case, but contrary to the condition studied by Schofield in which the skin web extended more distally on the right than on the left foot. Webbing in the feet of the father, *B* II-9, and the uncle, *B* II-11, of the propositus was similar to webbing in the feet of the latter. In Heston's two zygodactylous families the extent of webbing in the different affected members was highly variable.

Figure 12*B* shows a diagram of pedigree *B*. The authors are indebted to Messrs. William Kearney and Roy Drewett, former genetics students of the second author, for this pedigree. The second author is acquainted with the propositus, *B* III-12, and can vouch for the authenticity of the pedigree. Webbed toes in pedigree *B* are transmitted for three generations in the male line as in Schofield's case. With the exception of two sons who died in infancy without any record as to whether or not they possessed webbed toes, all sons of *B* I-1 showed the defect as well as the only son of *B* II-9. Sons of the daughters of *B* I-1 were normal as were all women in the pedigree. Previous authors agree with Schofield that his webbed toes pedigree shows *Y* chromosome inheritance of the abnormality. For example, Snyder states, "not all types (i.e., webbed toes) are inherited in this way (e.g., on the *Y* chromosome), but in Schofield's pedigree the condition involving the second and third toes was passed on from affected fathers to all their sons for four generations, fourteen affected males being included in the pedigree." Pedigree *B* is so short that it is possible that webbed toes in this pedigree do not differ genetically from the other cases of webbed toes already discussed. If so, crossing over does not take place in pedigree *B*.

The authors are indebted to the Medical Department of the Marine Training Command, Camp Joseph H. Pendleton, for the X-ray plate for pedigree *A*; to the Marine Photographic Section, for photographic reproduction of the X-ray plate and photograph Figure 11*A;* and and to Mr. Kenneth Matthews, Physics Department, North Texas Agricultural College, for the photograph Figure 11*B*.

Summary

Two new pedigrees of zygodactyly are described. Application of Haldane's method to the authors' pedigree *A* and to six other pedigrees of webbed toes suggests that zygodactyly in these cases is dependent upon a dominant partially sex-linked gene.

Literature Cited

1. Castle, W. E. *Jour. Hered.* 14:209-210. 1923.
2. Haldane, J. B. S. *Ann. Eugen.* Lond., 7:28-57. 1936.
3. Heston, W. E. *Jour. Hered.* 23:379-381. 1932.
4. Hurlin, Ralph G. *Jour. Hered.* 11: 334-335. 1920.
5. Schofield, Richard. *Jour. Hered.* 12: 400-401. 1921.
6. Schultz, Adolph H. *Jour. Hered.* 13: 113-117. 1922.
7. Snyder, Laurence H. *Medical Genetics.* Duke University Press. 1941.
8. Wright, Sewall. *Jour. Hered.* 13:118-119. 1922.

Table 1. Crossover and non-crossover progeny of thirteen critical marriages.

Male spouse of critical marriage		Offspring			
		Same sex as affected paternal grandparent		Opposite sex as affected paternal grandparent	
		Affected	Not affected	Affected	Not affected
The Pipkins	A II-1	1	2	0	0
	A III-3	0	0	0	3
Heston --	A II-2	1	0	1	0
	A II-3	2	1	2	1
Heston --	B II-1	3	2	0	1
	B II-3	1	1	0	3
Hurlin --	II-1	2	1	1	2
Wright --	II-5	0	0	1	1
Pfitzner (Schultz) -	III-1	1	0	2	0
	III-3	4	0	0	2
	III-6	0	0	0	2
	III-7	0	0	0	2
Wolff (Schultz) -	I-5	2	1	0	0
Total		17 non-co	5 co	7 co	17 non-co
Grand total : 34 non-crossovers; 12 crossovers. Crossover percentage : 30.6.					

FUSED TEATS

A Hereditary Defect in Beef Cattle*

LESLIE E. JOHNSON
South Dakota Agricultural Experiment Station

A TEAT defect in which the fore and rear teats were grown together was observed in the Hereford herd at South Dakota State College. This abnormality was so striking, and so undesirable, that an investigation was started in 1941 to determine its extent within the herd and its probable mode of inheritance.

Description†

This defect can best be described as a teat fusion. It appears to have its origin in the faulty placement of the fore and rear teats. The placement locates the teats in apposition at their bases on each side of the udder. This arrangement seems to create the fusion.

The fused condition occurred on either the right side or the left, but was never observed on both sides of any one udder. In all females where the teats on one side of the udder were fused, those on the other side were located extremely close together. Also, there were no cases in which both pairs of teats were extremely close without some fusion on one side. The fused condition extended from one-third to three-fourths the length of the teats in the animals examined. Palpation of the udders and teats indicated the fused condition involved the skin only, the walls of the lactiferous sinus being completely separate for each teat. The ends of the fused teats and teat canals appeared normal in all cases. Milk could readily be forced from either or both of the streak canals by proper application of pressure. Plain squeezing, however, often resulted in removing the milk from only one of the two quarters. The calves had no difficulty in getting all the milk from any of the cows. The right and left sides of the udders of two cows possessing the defect are shown in Figure 13*B* and *D*.

Occurrence

In 1941, when the author first observed the defect, there were sixteen females in the herd that were one year of age or older. Five of these possessed the defect. Four of the fused teats were on the right side of the udders and one was on the left. The cow with the defect on the left had a full sister with defective right teats. The pedigree chart shown in Figure 14 gives the parentage and distribution of normal and defective cows in the herd up to and including 1944.

All of the male calves born in the herd since 1941 were examined. The fused condition was not found in the rudimentary teats of any of these males. This, however, does not mean that the defect is limited to females, as all sires which produced the defective females had left the herd previous to that date.

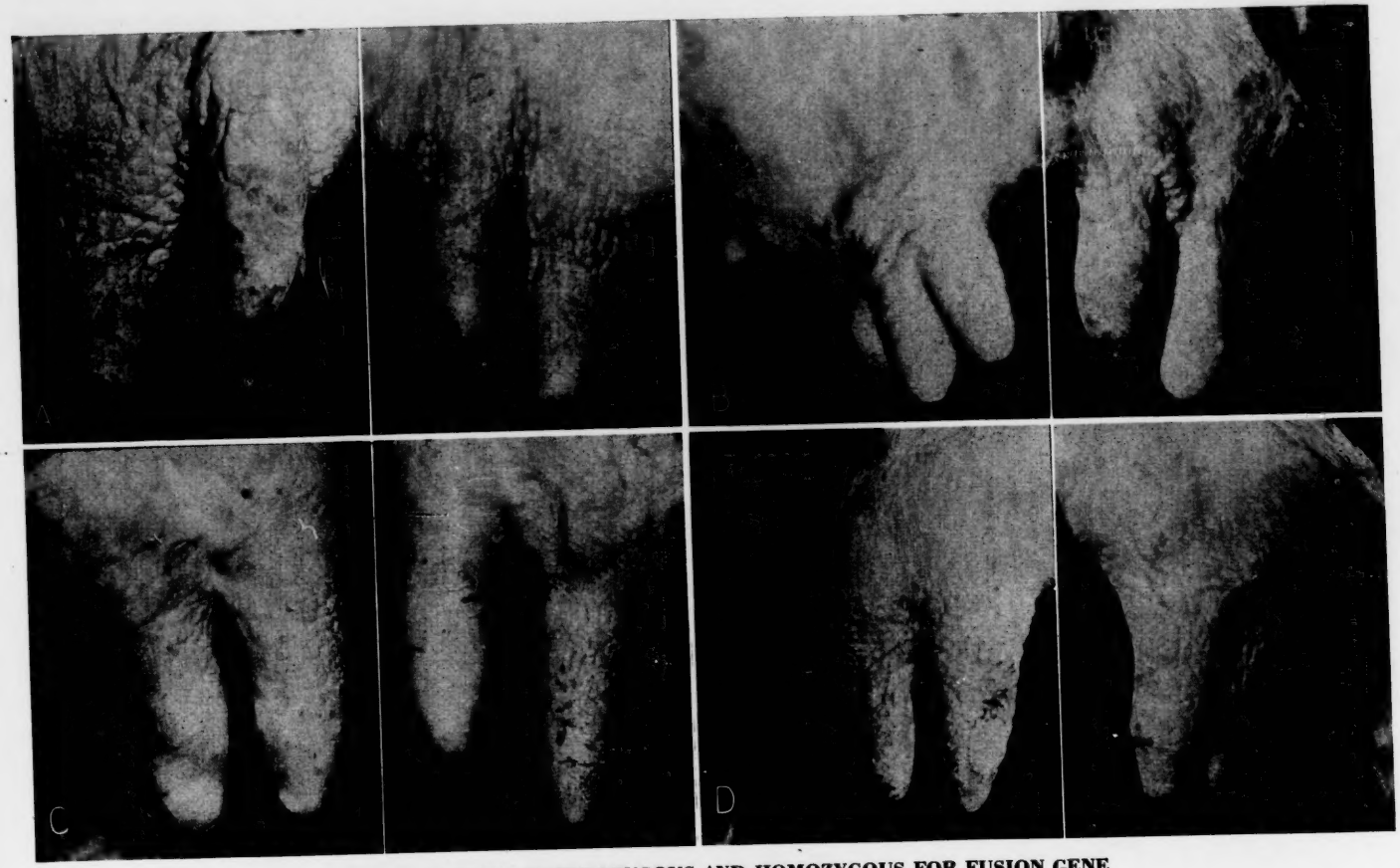
The sixteen cows in the herd in 1941 did not represent an unculled population. Also no records were available on the occurrence of the defect in the cows that had previously left the herd. Due to the fact, however, that an attempt was being made to expand the herd and that culling was based primarily on productivity and type, it is doubtful if the ratio of normal to defective animals was greatly altered through culling.

Breeding System Followed

During the period when the defective cows appeared the breeding system followed in the herd was that of rather intensive linebreeding. The linebreeding

*Approved for publication by the Director of the South Dakota Agricultural Experiment Station, Brookings, South Dakota, as Journal Series No. 197.

†The author is indebted to Dr. G. S. Harshfield, Head S. D. S. C. Veterinary Department. for his collaboration in studying the extent and nature of the defect.



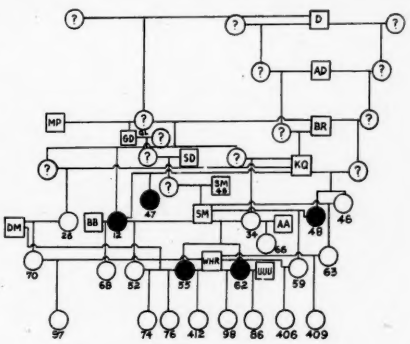
UDDERS OF COWS HETEROZYGOUS AND HOMOZYGOUS FOR FUSION GENE

Figure 13

A and *C* show right and left sides of udders of cows 34 and 52. These udders, with the teats abnormally spaced, are typical of cows heterozygous for the defective gene. Cow 34 produced two daughters (55 and 62) with the defective fused teat condition. *B* and *D* show right and left sides of udders of cows 47 and 55. These cows exhibit the two extremes of the fused condition found in the animals studied. According to the theory of inheritance proposed, these dams are homozygous for the recessive defective gene.

was directed chiefly toward the sire *BR*, an outstanding show and breeding bull. *BR* was purchased in 1924 and for thirteen years he and his son, *KQ*, sired all the offspring in the herd. The sire *SM* followed *KQ*. *SM* was a great, great, grandson of the cow *QL*. *QL* had many descendants in the herd at the time *SM* was used. This is plainly shown in the pedigree chart. The three bulls, *BR*, *KQ*, and *SM*, each sired defective individuals. The following five bulls that were used in the herd (all of which were unrelated to the preceding three) sired only normal animals.

During the time the sires *BR*, *KQ*, and *SM* were used, the inbreeding increased from four per cent to eight per cent. This is a considerably greater rate of inbreeding than is found in the breed as a whole. It is great enough to result in a rapid increase of a rare recessive gene present within the herd. Willham[1] reported an average increase of inbreeding in the Hereford breed of only .6 per cent per generation, and a value of 8.8 per cent for the *inter se* relationship of the whole breed. The *inter se* relationship in the herd following the use of sire *SM* was 33.4 per cent. The sire *BR* had an average relationship coefficient to the herd of 37.7 per cent in 1938 following the use of *SM*. *BR*'s son, *KQ*, had an average relationship of 43.[illegible] per cent to the herd at that time. The dam *KL*, the great, great granddam of *SM*, had an average relationship of 21.4 per cent, thus being almost the grandmother of the entire herd in 1938. In 1944, she still had a relationship of 19.8 per cent to the herd. As shown in the pedigree chart, *QL* and *BR* must both have possessed the defective gene or genes.



PEDIGREE CHART OF HERD

Figure 14

Females represented by solid symbols possessed the fused teat defect. Those represented by hollow symbols were normal. A "?" within a symbol indicates that the female was unobserved for the defect. The only males shown in this chart that were examined were sires UUU and WHR. These showed no fused condition of the rudimentary teats.

Mode of Inheritance

The frequent occurrence of the defect during the period the related sires *BR*, *KQ*, and *SM* were used, and its complete absence during the immediately succeeding period in which only unrelated sires were used, seems to establish the heritability of the fused teat condition. The number and distribution of defective animals suggest that the fused condition was inherited as a simple monofactorial recessive. The number of animals involved in the different matings, however, was too small to furnish significant statistical proof of this hypothesis.

Although the defect, "fused teat," seems to have occurred only in animals homozygous for the recessive gene, the heterozygotes did not appear to have a normal space between the teats. The data on teat placement were somewhat obscured by variation in age, size, and stage of lactation, and by the general lack of uniformity in beef cattle udders, but there was a definite gradation between strictly normal and defective cows. If the defect is inherited as a simple monofactorial recessive, dams 34, 52, 59, 68, 76, 86, 98, and 412 are heterozygous for the defective gene. Classifying the distance between teats on the sides of the udders of these cows on a scale consisting of normal, intermediate, and defective, there were seven intermediate placements of teats and one normal placement. Thus, it appears the heterozygotes are largely of the intermediate type as far as teat placement is concerned. Cows 63 and 66 both possessed normal teat placement indicating

they were of the homozygous normal genotype. If the defect is inherited as a simple Mendelian recessive this is possible, for the pedigree chart indicates that one cow had a normal and a heterozygous parent; and the other, two heterozygous parents or one heterozygous and one normal parent. The right and left sides of the udders of cows 34 and 52 are shown in Figure 13.

Summary

A defect, "fused teat," was observed in a herd of Hereford beef cattle. It appeared to result from the teats being located in apposition at their bases on the sides of the udder. The defect occurred on either the right or left side, but never on both sides of one udder. The fusion extended from one-third to three-fourths the length of the teats.

The frequency and distribution of the defect indicated it was inherited as a simple monofactorial recessive. Dominance, however, existed only in the sense that the heterozygotes possessed no teat fusion. In teat placement, the udders of heterozygous females were intermediate between the normal and the "fused teat" condition.

Literature Cited

1. Willham, Oliver S. *Jour. Hered.* 28: 283-294. 1937.

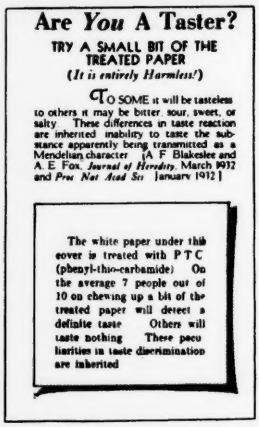
"WITHOUT PRIDE OF ANCESTRY"

The Decision of Judge Oliver in the Celebrated Jackass Case *(121 Neb. App. 72)*, which should be in the genetic "Record."

THE evidence before the Court is amply sufficient to establish an express warranty on the part of the defendant that the animal in question was a fit and suitable one for breeding purposes, and the Court therefore finds that there was a warranty. But even conceding that there was no such warranty, there surely can be no question, under the evidence, that there was an implied warranty as to its fitness as a breeder and foal-getter. The defendant must surely have known that the plaintiff was purchasing the animal for breeding purposes only. He knew at the time of making the sale that no reasonable man would attempt to use a jackass for any other purpose than to outrage nature by propagating mules. He could not have supposed that the plaintiff desired to acquire a jackass for a pet. The animal is wholly unsuitable for that purpose. Its form is neither pleasing to the eye, nor its voice soothing to the ear. He is neither ornamental in his appearances, nor amusing in his habits; he is valuable only as he is able and willing to propagate the mule species.

It appears from the evidence that after purchasing the animal, the plaintiff on several occasions caused him to be placed in the society of certain soft-eyed, sleek-coated young mares, that were in the pink of that condition which is supposed to arouse the interest and attract the attention of any reasonably amorous jackass, but that he passed them up and knew them not. The defendant admits representing to the plaintiff that the jackass in question would do that work for himself. But the evidence shows that if he was ever possessed of that valuable and charming accomplishment, he failed on the occasions just mentioned to practice it with the zeal and ardor becoming of an ambitious jackass in full possession of his faculties.

He was, indeed, a worthless, unpedigreed and impotent jackass, without pride of ancestry or hope of posterity—a source of disappointment to his female friends, and an item of expense to his owner. There is no brute in all the animal kingdom more worthless than a Missouri-bred jackass afflicted with lost manhood. He was not as represented and warranted by the defendant, and the plaintiff is entitled to recover.


Are You A Taster?
TRY A SMALL BIT OF THE TREATED PAPER
(It is entirely Harmless!)
TO SOME it will be tasteless to others it may be bitter sour, sweet, or salty These differences in taste reaction are inherited inability to taste the substance apparently being transmitted as a Mendelian character [A F Blakeslee and A E Fox, Journal of Heredity, March 1932 and Proc Nat Acad Sci January 1932]
The white paper under this cover is treated with PTC (phenyl-thio-carbamide) On the average 7 people out of 10 on chewing up a bit of the treated paper will detect a definite taste Others will taste nothing These peculiarities in taste discrimination are inherited

LEAFLETS TO DEMONSTRATE HEREDITY

Last year the "Taster test" served as an introduction to human genetic differences in many classrooms. The test is so easily made with the treated paper, and arouses such great interest that it appears to have a definite place in group demonstrations of such differences. These leaflets, size 3⅜" by 5½", are available in quantities for those who wish to use them for classroom or lecture purposes. On the back is printed an outline 4-generation pedigree chart. Sample on request. Prices: 10 for 25c; 50 for $1.00.

AMERICAN GENETIC ASSOCIATION

1507 M STREET, N. W. WASHINGTON 5, D. C.

FEEDS for Your "Lab" Animals

DEPENDABLE feeds are important in the laboratory where tests of various kinds are being made on small animals.

Dickinson, producer of the well known Globe Feeds, is one of the oldest and most reliable feed houses from which to buy your "Lab" animal feed.

Globe Feeds supply complete rations. They are dry . . . easily stored . . . take up little space . . . and involve little waste. They hold the same high standard of quality from year to year.

GENEROUS SAMPLES FREE

To supervisors of experimental animals, we will send a free sample, large enough to make an adequate test. Just tell us the kind and quantity of food desired.

FOR RABBITS

Globe Rabbit Glob-ets to be supplemented with hay.
Globe Rabbit with Alfalfa, meal form—complete.
Globe Rabbit with Alfalfa, pellet form—complete.

FOR RATS AND MICE

Dickinson's Dog Food—Nugget—Pellet or meal form. Dickinson's Kibbled Dog Food.

FOR GUINEA PIGS

Globe Rabbit Glob-ets to be supplemented with hay.
Globe Rabbit with Alfalfa, meal form—complete.
Globe Rabbit with Alfalfa, pellet form—complete.
Supplement with Vitamin C carrier three times a week.

THE ALBERT DICKINSON COMPANY

2714 WEST 35TH STREET CHICAGO 90, ILLINOIS

A new text in elementary genetics
by an eminent geneticist

GENETICS

EDGAR ALTENBURG
Rice Institute

Over twenty-five years of classroom experience have convinced Professor Altenburg that a sound knowledge of the physical basis of heredity is absolutely essential to an understanding of genetics. In this text the student is made aware, from the beginning, of the important role played by genes and chromosomes.

Fundamentals are covered in an unusually clear and comprehensive fashion, with the emphasis being placed on *principles* rather than on details of a highly technical nature. Each concept and phase of development is explained patiently and in detail on a level with the beginner's interests and understanding.

Completely modern in its approach, the book includes wherever possible the newest and most important developments in genetic research. Indicative of the thoroughness with which Altenburg has covered this new material are the complete chapter on cytogenetic maps and his inclusion, in the discussions on sex determination, of the work of Whiting, Winge, and Sonneborn.

Comprehensive summaries and numerous problems and exercises follow each chapter to aid in study and provide material for discussion and special assignments. Fully illustrated with 148 diagrams, drawings, and photographs.

452 pages **PUBLISHED JULY 1945** **$3.20**

HENRY HOLT AND COMPANY, New York 10